OBSTETRIC EMERGENCIES

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DRUG DOSAGE

The authors and publisher have exerted every effort to ensure that drug selection and dosage set forth in this text are in accord with current recommendations and practice at the time of publication However, in view of ongoing research changes in government regulations and the constant flow of information relating to drug therapy and drug reactions the reader is urged to check the package insert for each drug for any change in indications and dosage and for added warnings and precautions This is particularly important when the recommended agent is a new and/or infrequently employed drug

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Preface

These will enable the obstetrician to decide which is "watchful waiting which is 'hopeful procrastination" and which is "criminal negligence"

W J Dieckmann
Am J Obstet Gynecol
50.590, 1945

A preventable maternal death is the greatest tragedy in medical practice Obstetric emergencies allow little time for thought, and often only the prompt and appropriate intervention of an alert doctor or nurse averts catastrophe This book is written specifically to provide guidance to specialists, residents, family practitioners, and nurses who are involved in the management of the acutely ill obstetric patient.

Because of the apparent interest in the subject a monograph was written in 1961 The theme has been expanded in this edition to include emergencies of all types which may be met with during pregnancy, labor and the immediate postpartum period Detailed consideration is given to such areas as clotting disorders, life threatening infections, shock, acute renal failure anesthetic emergencies and the use of ultrasonography Throughout the book an effort is made to present the reader with a positive approach to emergency situations Where possible, a definite course of action is outlined, and modern trends in diagnosis and management are stressed. Drug dosages suitable for the "average" patient in each particular situation have been given but the importance of individualization is stressed

This is not an obstetric textbook but rather a "ready reference manual' on obstetric emergencies With this in mind a serious effort has been made to provide a logical chapter sequence a detailed table of contents and a good index Where necessary the text is supplemented with figures and tables For their assistance with these we thank the staff of the Departments of Medical Illustration of the University of Miami School of Medicine and St Louis University The original illustrations are mainly the work of Mr William McNab, to whom we are grateful for his many recommendations

For numerous helpful suggestions we owe thanks to our colleagues Dr James M Ingram, Dr J Donald Wargo, Dr Paul E. Demick, Dr Allan McLeod Dr Edward J Diamond Dr H Praphat, Dr Manuel R Comas, and to Sister Jeanne Meurer, M.S, C.N.M, of St Louis University School of Nursing For their assistance in typing the manuscript we thank Barbara Chorley, Moira Morgan and Rita Florentino

For their help and patience throughout the preparation of the manuscript we thank the staff of Harper & Row

Finally, this book is dedicated to mothers everywhere and especially to Margaret, Jane, and Mary

D C.
R.E.W.
T C F O C.

Common Obstetric Emergencies

Chapter 1

Ralph E. Woods, Denis Cavanagh

There are but two things that have much effect on me at a labour haemorrhage and convulsions"
William Hunter (1718–1783)

In the world today fewer than 1% of pregnant women are under the care of specialist obstetricians Necessity dictates that most women are delivered by midwives, handywomen, and husbands In quality, the midwives vary from the neat well trained American or European nurse to the unkempt, incantation-chanting crones who play the role in many other parts of the world

Despite statements to the contrary, based largely on failure to recognize that different countries define perinatal mortality differently, the standard of perinatal care in the United States of America is among the highest in the world About 95% of pregnant women see a physician at some time during pregnancy Although only about 50% of these are under the care of a specialist obstetrician, there is little hazard in this provided 1) the patients receive good antepartum care, 2) they are referred for specialist consultation should any abnormality be detected, and 3) they are delivered in an adequately equipped hospital

The physician who practices in an area with available specialists will usually be able to refer his high risk patients without difficulty On the other hand, a rural family practitioner may have no specialist consultation available and less than adequate hospital facilities Although the latter situation is an undesirable one, it will not be made to disappear simply by ignoring its existence Many women's lives are saved annually by the family physicians and nurse midwives on our urban and rural medical frontiers Every effort must be made to give them as much help as possible while plans go forward to provide the needed facilities for specialist consultation, transfusion, and major surgery in these areas

Even in low-risk patients, obstetric emergencies may develop, so all doctors and nurses who participate in the care of pregnant women must be prepared to deal with such situations

To plan this book along practical lines, an effort was made

to determine the types of emergencies most commonly met with in home ·' ' ies These statistics are available in the reports of obstetrical emergency ser· set up in areas where domiciliary obstetrics is common A physician a small hospital will be confronted with similar situations Thus an analysis · these emergency service calls brings the problem into better perspective than ar review of serious complications from a large obstetrics department

There has recently been a trend for "consumers" to demand home deliver in the United States, so here as elsewhere we must be prepared to cope wi emergencies in the home setting Current statistics on obstetric emergenci are difficult to obtain However, there is no reason to believe that the ord of frequency has altered significantly in the past 25 years so far as matern emergencies are concerned

Collected figures representing emergencies occurring under diverse circum stances in both rural and urban areas were reviewed (Table 1-1) Obviousl hemorrhage and convulsions are the bugbears of obstetric practice, and whe we add sepsis we have the three most common causes of maternal deatl Many of the emergencies could be avoided by better antepartum care, bu there is no doubt that unforeseen complications will arise despite the mo careful screening

In the last several decades, policies aimed at universal hospital confinemer have been promoted in the interest of mother and infant Acceptance and im plementation of these policies have been associated with dramatic declines i maternal and perinatal mortality Although there is no certainty that these re lationships are causally related, it remains our firm belief that in the absenc of economic pressure and geographic necessity, home deliveries or deliverie in inadequately equipped and staffed facilities are earmarks of an inefficien maternity and newborn care system When emergency situations occur, eve in low-risk patients, time is never on the side of the patient

In view of increasing consumer demands to return pregnancy, labor, de livery, and newborn care to a home-, family-, and parent-controlled environ ment, there may be today an even greater need to reidentify complications an emergency situations in mothers and their infants and to reemphasize principle of management This is particularly true when practitioners—physicians o midwives—attend too few patients to maintain competence in the recognitior and management of these complications

Major social changes have occurred in the area of human reproduction including 1) decline in domiciliary care, 2) decline in the birth rate in many areas, and 3) the shifting age parity distribution of births These and othe changes have undoubtedly affected the incidence of certain complications Current statistics on similar populations served by similar systems are difficul to come by, but as already stated, there is no reason to believe that the order of frequency of maternal complications has been altered in the past 25 years

There is need, however, to draw attention to the increasing variety of com- lications that might be encountered in the delivery of maternity and new- infant care These are the result of the changing nature of obstetrics The changing physical and medical characteristics of women undertaking child- birth account for some Others are due to improved recognition and under- standing of pathologic and pathophysiological entities Some might be con idered as iatrogenic—i e, arising as a result of certain practices utilized in and neonatal care Lastly, certain emergencies and complications

TABLE 1-1. Common Obstetric Emergencies

Type of Emergency	Dewhurst (1952)	Hagberg (1956)	Sutherland (1959)	Total	%
Postpartum hemorrhage and retained placenta	245	65	71	381	45 1
Postpartum hemorrhage and shock	143	102	44	289	34 2
Abortion	33	3	14	50	5 9
Eclampsia	23	4	6	33	3 9
Antepartum hemorrhage	9	14	10	33	3 9
Secondary postpartum *hemorrhage*	9	3	3	15	1 8
Other conditions	27	1	16	44	5 2
Total	489	192	164	845	100 0

(Dewhurst CJ Emergency *obstetrical service review of 489 cases in Manchester area* Lancet 2 746, 1952, Hagberg CJ The Capetown obstetric flying squad Its conception, *organization and operation* South *Afr Med J* 30 1140 1956, Sutherland AM Personal communication recorded flying cases, Southern General Hospital, Glasgow, Scotland, April 15, 1956—July 20, 1959)

TABLE 1-2. Some Types of Complications in Domiciliary Cases

	Patients	
Condition	No.*	No.†
Total No of Patients	287	155
Normal deliveries	231	133
Patients with complications	56 (19%)	22 (14%)
Toxemia	1	6
Hemorrhage	3	1
Prolonged labor	31	15
Malpresentations	1	2
Prolonged *rupture of membranes*	7	—
Induction/augmentation	0	14
Lacerations	53	—
Infections	5	—
Mothers transferred to hospitals	56	3
Mothers needing C-section	4	0
Prematurity	8	1
Fetal distress (meconium/bradycardia)	6	9
Early respiratory problems	9	29
Stillbirth	1	0

(Adapted from *Mehl LE et al.: Birth and Family 2·4, 123, 1975, †Cox CA et al Br Med J 1 84, 1976)

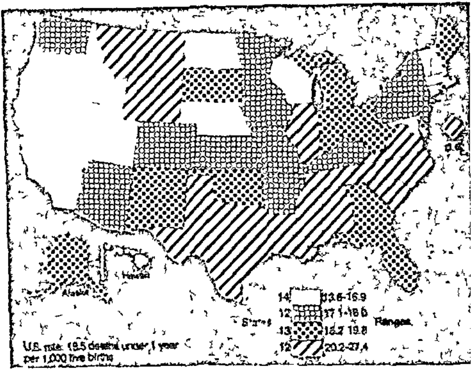
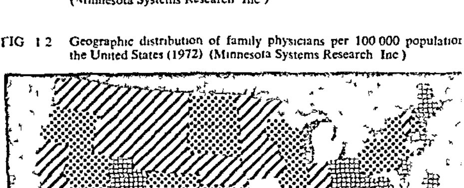


FIG 1 1 Geographic distribution of infant mortality in the United States (19
(Minnesota Systems Research Inc)

FIG 1 2 Geographic distribution of family physicians per 100 000 populatior
the United States (1972) (Minnesota Systems Research Inc)



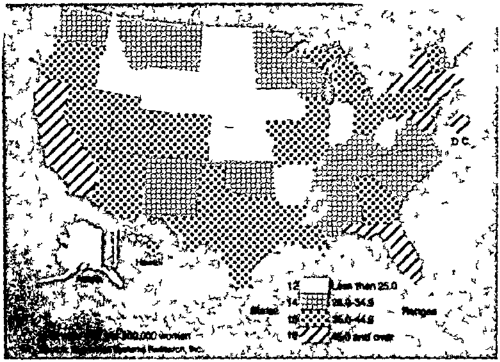
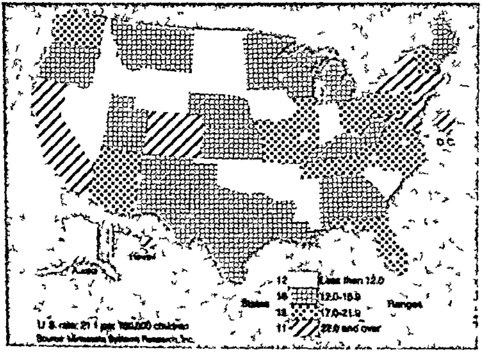


FIG 1 3 Geographic distribution of obstetrician gynecologists per 100 000 women aged 15–44 in the United States (1972) (Minnesota Systems Research Inc.)

FIG 1-4 Geographic distribution of pediatricians per 100 000 children under 20 years of age in the United States (1972) (Minnesota Systems Research Inc)



(Table 1-2) in the newborn are presented inasmuch as those who atten labor and delivery are often responsible for the immediate care of the newborn It is emphasized that the two populations in which the complications wer observed are not comparable in number, time, social, or other characteristic

If an indicator of maternal and infant care, such as infant mortality, is con sidered, it will be noted that in the United States there is a considerable range depending upon geographic location and the population served (Fig 1-1) It will also be noted that the availability of an adequate number of providers c care is related to the improvement in infant mortality rates (Fig 1-2 to 1-4)

Further reduction in maternal and perinatal mortality and morbidity will b reached only through the combined efforts of consumers, providers, an government Improvement in physical and emotional health, social circum stances and patterns of childbearing have an important role to play Wide utilization of improved antepartum, intrapartum, and neonatal care is obvi ously important, particularly in the context of tiered, regional perinatal pro grams The need for emergency care can be expected to decrease as the genera standard of maternal and infant care improves However, the provider— whether obstetrician, nurse, midwife or other—must always be prepared t meet the unheralded emergency if tragedy is to be averted

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Chapter 2

Clotting Disorders in Pregnancy

Denis Cavanagh

there are alterations in the blood or blood vessels of a temporary nature which prevent its clotting and thus during labor or operations cause death "

J B de Lee

American Journal of Obstetrics 44 785 1901

Pregnancy predisposes women to certain clotting disorders, and their occurrence during pregnancy may in turn predispose to hemorrhage, shock, and even maternal and fetal death For this reason, the obstetric team must be familiar with some of the more common coagulation disorders that may be encountered during pregnancy, labor and the puerperium

REVIEW OF HEMOSTASIS

To understand these clotting disorders properly, knowledge of the normal mechanism of hemostasis is necessary Hemostasis is the general term applied to the life saving process that stops the flow of blood from injured vessels Certain requirements must be met 1) blood vessels and extravascular tissues must be normal, 2) platelets must be normal in quantity and quality, and 3) clotting factors in plasma and serum must be present in adequate quantity (Table 2-1)

THE THROMBOTIC PROCESS

The coagulation process may be divided into three phases the vascular phase, the platelet phase, and the coagulation phase

The Vascular Phase

When an injury occurs to a small vessel and results in the extravasation of blood, three responses follow that help limit the injury 1) Vasoconstriction markedly reduces blood flow

TABLE 2-1 *Coagulation Factors Present in Plasma and Serum

Factors present in plasma	Normal values (*mg/100 ml*)	Presence or absence in *normal serum*
*I Fibrinogen	200–400	Absent
II Prothrombin	75–125	5%
V *Labile factor proaccelerin*	75–125	Absent
VII Stable factor proconvertin	75–125	Present
VIII Antihemophilic globulin	50–200	Absent
IX Plasma thromboplastin component	50–200	Present
X *Stuart Prower factor*	*75–125*	*Present*
XI Plasma thromboplastin antecedent	70–130	Present
XII Hageman factor	70–130	Present
XIII Fibrin-stabilizing factor		
Fibrinase	*50–200*	*Absent*
Platelets	150 000–400 000/mm³	Absent

*Roman numerals indicate standard or international nomenclature for coagulation factors Missing are III thromboplastin a tissue factor not normally present in circulating plasma or *serum in significant amounts IV calcium ion present in all tissues and fluids and VI not included in current nomenclature*

(Cavanagh D Comas MR In Romney S Gray MJ Little B et al [eds] Gynecology and Obstetrics The Health Care of Women New York, McGraw Hill Copyright © 1975 Used with permission)

through the area 2) The escape of blood into the relatively rigid extravascular supporting tissue increases the pressure within these structures and helps collapse capillaries and venules 3) Various substances such as tissue *thromboplastin* (factor III) and adenosine diphosphate (ADP) are released from the injured tissue, *and these help to initiate* the final coagulation phase

The Platelet Phase

Platelets adhere to the surface of the injured vessel and aggregate to one another almost instantaneously after an injury This results in the formation of a platelet plug that may provide temporary or complete hemostasis, depending on the extent of the *injury Platelet adhesion and aggregation* are encouraged by ADP (derived from injured tissues) and the anti-VW *factor (so named* because of its lack in Von Willebrand s disease)

Platelets have other functions in normal coagulation They release adenosine triphosphate (ATP) as well as ADP and various phospholipids *collectively* known as platelet factor III They also release thrombosthenin, a contractile protein that is responsible for the clot retraction

The Coagulation Phase

This phase is absolutely necessary for the formation of a firm thrombus, which will later be the structural basis for the reconstructive process

The mechanisms by which the coagulation factors interact is not exactly

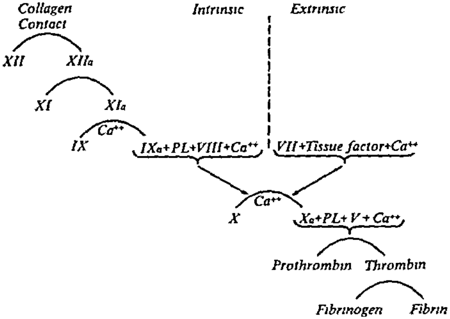


FIG 2 1 Cascade diagram, showinc scheme of intrinsic and extrinsic blood coagulation PL, platelet factor 3 or phospholipid (Cavanagh D, Comas MR In Romney S, Gray MJ, Little B, *et al* [eds] Gynecology and Obstretrics The Health Care of Women New York, McGraw Hill, Copyright © 1975 Used with permission)

known, but when activated, each factor sequentially activates the factor next in line in a "waterfall" sequence This is the so-called "cascade theory" of coagulation referred to by Macfarlane and by Davie and Ratnoff The cascade is accomplished by two pathways (Fig 2-1)

THE EXTRINSIC PATHWAY This involves a rapid process in which the procoagulant material comes from the tissues and is not ordinarily present in the bloodstream

THE INTRINSIC PATHWAY This is a slower process, in which the coagulation proteins, which normally circulate in inactive form, are sequentially activated The end result of both pathways is the deposition of fibrin

The coagulation phase begins with the phenomenon of surface activation of factor XII (Hageman) and involves the conversion of factor XI to its activated form XIa, sometimes called plasma thromboplastin antecedent *In vivo*, factor XII is activated by contact with skin or any collagenous structure *In vitro* it occurs when it is exposed to electronegative surfaces such as glass

The activation of factor XI initiates the activation of factors IX (plasma thromboplastin component) and VIII (antihemophilic globulin) Together with calcium and platelet factor III, factors IX and VIII are involved in the conversion of factor X into its enzymatic form Factor Xa forms a particulate complex (prothrombinase), with factor V (accelerator globulin), calcium, and platelet phospholipid Prothrombinase then initiates the conversion of prothrombin into thrombin

A functionally identical prothrombinase can be produced in a matter of seconds by activation of the extrinsic pathway This pathway bypasses the activation of factors XII, XI, IX, and VIII Tissue thromboplastins released by injury interact with calcium and factor VII (serum prothrombin conversion accelerator), and the resulting complex causes activation of factors X, V, an platelet phospholipid into prothrombinase

In the final sequence of the coagulation process (Fig. 2-2), under th effects of the prothrombinase complex, factor II (prothrombin) is activate to thrombin This then reacts with factor I (fibrinogen) in the following mar ner Four peptides are split off from fibrinogen, leaving only fibrin monomer These monomers and peptides form a visible but unstable fibrin polym (soluble fibrin) Finally, factor XIIIa (fibrin-stabilizing factor) brings abo the formation of a stable fibrin polymer (insoluble fibrin) This insoluble fibr gives rise to the permanent hemostatic plug The last stage in the coagulatic process is that of clot retraction, which may help bring together the margins the injury This takes place under the influence of a contractile prote (thrombosthenin) and ATP supplied by the platelets

Although the clinical situation is assessed by means of *in vitro* testing the are important differences between intravascular clotting and clotting in t test tube Even in a patient dying from hemorrhage associated with coag lopathy, there is depletion but never a total lack of all factors, because an *vivo* negative feedback mechanism accelerates the production of deplet factors as much as possible However, when blood clots in the test tube, t reaction proceeds to completion and the activity ceases

THROMBOLYSIS

Normally, the formation of fibrin is not a continuous process The body h ways of limiting the coagulation process so that the clot does not propag beyond the site of the wound There are proteolytic enzymes in tissue a leukocytes that act as antithrombins, antithromboplastins, and inhibitors prothrombinase The reticuloendothelial system and liver macrophages h remove from the circulation some of the activated coagulation factors

The fibrinolytic system is usually considered the major physiologic mea of disposing of fibrin after hemostasis has been secured (Fig 2-3) Fibrinoly can be carried out by leukocytes but most of it is carried out by the activati of a potent plasma protease called plasmin This exists in normal plasma the form of an inactive precursor, plasminogen, which can be activated hypoxia, tissue extracts, bacterial enzymes (streptokinase and urokinase), ac vated factor XII, thrombin, and hypoglycemia

Usually plasminogen is deposited within the clot during the clotting proce When activating substances from plasma, blood vessels or tissues penetrate t clot, plasminogen is converted into its active form, bound plasmin, and localiz secondary fibrinolysis ensues.

Normally antiplasmins in plasma rapidly destroy free plasmin but are i effective against bound plasmin, which is thus available to carry out its phys logic function Plasmin splits fibrin and fibrinogen into progressively small fragments (X, Y, D, and E), which are referred to as fibrinolytic split produc (FSP), fibrinolytic degradation products (FDP), or fibrin(ogen) related an gen (FRA) The fragments of fibrin and fibrinogen vary in their sensitiv to fibrinolysis Normally, minimal amounts of FSP are found in the circulatio

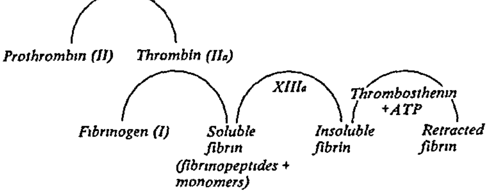
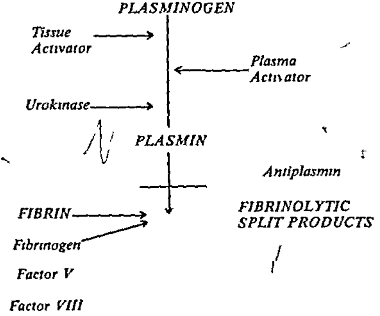


FIG 2 2 Final sequence of coagulation process (Cavanagh D, Comas MR In Romney S Gray MJ, Little B *et al* [eds] Gynecology and Obstetrics The Health Care of Women New York McGraw Hill Copyright © 1975 Used with permission)

FIG 2 3 Activation mechanism of thrombolytic system (Cavanagh D, Comas MR In Romney S, Gray MJ Little B *et al* [eds] Gynecology and Obstetrics The Health Care of Women New York, McGraw Hill, Copyright © 1975 Used with permission)



When present in large quantities, FSP act as powerful anticoagulants, impairing the aggregation of platelets and also their release of phospholipids, ATP, and ADP The FSP also interfere with plasma thromboplastin formation, the thrombin fibrinogen reaction, and the polymerization of fibrin They form inactive complexes with fibrin monomers, which are known as soluble fibrin monomer complexes (SFMC)

Once plasminogen is converted to plasmin, this substance breaks down both fibrin and fibrinogen Although the removal of fibrin may be life-saving in the

presence of widespread intravascular coagulation, the simultaneous destruction of circulating fibrinogen places the patient in danger of death from hemorrhage

However, just as there are inhibitors of the clotting mechanism, there are inhibitors of the fibrinolytic mechanism Several factors such as plasminogen, antiplasmins, and activator substances can be measured to assess the degree of fibrinolysis A marked decrease in the plasminogen level and an increase in FSP suggests marked fibrinolytic activity FSP assays are now available on an emergency basis using commercially available FSP antibody-coated latex particles

The most commonly encountered clotting disorders are as follows disseminated intravascular coagulation (DIC), probably better described as intravascular coagulation-fibrinolysis (ICF), idiopathic thrombocytopenic purpura (ITP), Von Willebrand's disease, circulating anticoagulant antibody syndrome and factor XI deficiency

DISSEMINATED INTRAVASCULAR COAGULATION

Disseminated intravascular coagulation (DIC), more correctly termed intravascular coagulation—fibrinolysis (ICF), is an apparently paradoxical situation in which the thrombotic and fibrinolytic mechanisms are being initiated simultaneously, so that intravascular coagulation and also a hemorrhagic diathesis are present at the same time

This is at first a difficult concept to grasp There are *two apparent paradoxes in DIC* 1) The stimulation of the clotting mechanism results in a bleeding defect, 2) the treatment of this bleeding defect with an anticoagulant may be appropriate in some cases The hemostatic process functions in a remarkably interdependent manner The presence of activated coagulation factors serves to initiate the clot-dissolving or fibrinolytic mechanism, probably to ensure control of the clotting mechanism When the thrombotic and thrombolytic mechanisms are in balance, hemostasis is assured Normally both processes are limited to a local area of injury Through the action of a variety of stimuli however, these two processes can be triggered within the vascular bed In pregnant patients intravascular coagulation is almost always primary, and fibrinolysis is a secondary response The bleeding diathesis results from the consumption of coagulation factors, the pathologic activity of the fibrinolytic system, and the anticoagulant effects of some of the by-products generated by this process

Many synonyms for "DIC-like syndromes" are still commonly used Such terms as "hypofibrinogenemia," "afibrinogenemia," or even "defibrination syndrome" simply describe the consumption of factor I or fibrinogen The term **consumption coagulopathy** recognizes that more than one clotting factor is consumed in this defect and is therefore more accurate Fibrination syndrome another term formerly used places more emphasis on thrombosis than on the bleeding defect

The term **intravascular coagulation-fibrinolysis** (ICF) is more accurate than DIC in that it recognizes the presence of fibrinolysis as well as clotting In the literature today the terms disseminated intravascular coagulation (DIC) and intravascular coagulation fibrinolysis (ICF) are used interchangeably, the former being the term most commonly used in obstetrics

McKay, in 1965, introduced the concept of multiple causality resulting in a common end point when he described DIC as an "intermediary mechanism of disease which is not necessarily bound to a single disease entity, but may be responsible for much of the pathology, morbidity and mortality attributed to the antecedent disease" This is a very attractive concept Disseminated intravascular coagulation can be a rapidly progressive entity with the potential to cause great injury to vital organs On the other hand, the underlying disease process that stimulates the development of the consumption coagulopathy should not be ignored In obstetric and gynecologic patients, aggressive treatment of this primary disease brings the DIC under adequate control and usually saves the patient's life

STIMULI LIKELY TO INITIATE DIC

The list of potential causes has continued to lengthen since the mid 1950s, when research interest in this process began to grow These stimuli are classified today in general categories as follows infusion of tissue extracts, endothelial damage, anoxia, bacterial endotoxins, chemical and physical agents, hemolytic processes, immune reactions, and thrombocytopenia

The obstetric conditions that cause DIC are

1. Abruptio placentae
2. Endotoxic shock
3. Intrauterine dead fetus syndrome
4. Amniotic fluid (embolism) infusion
5. Eclamptogenic toxemia
6. Hemorrhagic shock
7. Saline-induced abortion
8. Hydatidiform mole
9. Ruptured uterus Septic abortions.

A very small number of cases have also been reported in which DIC has developed with acute fatty liver Consumption coagulopathy has also been reported in neonates whose mothers suffered from toxemia, abruptio placentae, and Rh isoimmunization

INCIDENCE

The incidence of DIC in obstetric patients is not precisely known Although often undetected, DIC is probably invariably associated with abruptio placentae and with cases of amniotic fluid infusion in which the patient survives the embolic episode It is seen less frequently with other diseases Clearly, DIC is an important entity, and as we learn more of the dynamic processes involved in the thrombotic and thrombolytic mechanisms, it seems likely that we will find our way to a better understanding of many pathologic conditions

PATHOPHYSIOLOGY OF DIC

In obstetric patients, the majority of cases of DIC are initiated by the entrance into the circulation of tissue extracts of fluids high in thromboplastin content This is the case in abruptio placentae, the intrauterine dead fetus syndrome, septic abortion, and amniotic fluid embolism A similar mechanism is also

probably operative in eclamptogenic toxemia, molar pregnancy, ruptured uterus, and saline-induced abortions The entrance of thromboplastic substances into the circulation causes activation of the extrinsic pathway In amniotic fluid embolism, the fluid has little thromboplastic activity, but it has a high content of collagenous cellular debris and other particulate matter, which may cause platelet aggregation and the activation of factor XII and the intrinsic pathway In hemorrhagic and septic shock there is endothelial damage and platelet aggregation that initiate DIC by activation of the intrinsic pathway

Once DIC is established, coagulation will proceed *in vivo* much as it does *in vitro* Initially, there exists a state of hypercoagulability, which eventually consumes platelets, fibrinogen, and factors V, VIII, and XIII, sometimes almost to the point of total depletion

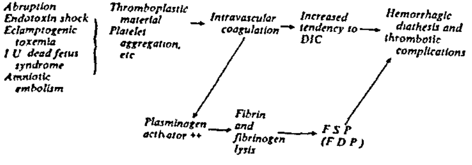
While the intravascular coagulation is going on, there is a tendency to thromboembolism, so the activation of the fibrinolytic system is a protective mechanism In DIC, plasmin is produced in quantities that exceed the capacity of the antiplasmins Circulating plasmin causes proteolysis of hemostatically important "fibrin" plugs, as well as of circulating fibrinogen in the presence of factors V and VIII, and results in the release of fibrin degradation products into the circulation

The end result of this process is a bleeding diathesis that complicates the primary illness, one that may be very severe in the presence of an open wound Several important clotting parameters are affected The platelet phase is impeded because of the thrombocytopenia Circulating plasmin lyses whatever fibrin may have been previously laid down at the wound's margin The generation of new fibrin is imperfect because of the depletion of the consumable factors and the presence of circulating anticoagulants (FDP) (Fig 2-4)

Hypercoagulability in Pregnancy

Does pregnancy produce a hypercoagulable state of the blood? There is still no agreement on what constitutes a hypercoagulable state Some investigators define it as a high level of coagulable factors in the blood, while others feel that the term must be used only to indicate those states that show an increased tendency toward intravascular coagulation of the disseminated type Pregnancy

FIG 2-4 Pathophysiology of DIC in obstetrics



fits either of these definitions because several coagulation factors increase during pregnancy, and DIC is certainly more prevalent in the pregnant than in the nonpregnant woman

Certain changes occur in the coagulation mechanism during pregnancy There are increases in fibrinogen, prothrombin, and factors VII, VIII, IX, and X Fibrinolytic inhibitors tend to augment the potential for intravascular coagulation Decreased concentrations of factors XI and XIII may help to counteract this augmentation Fibrinolytic activity falls steadily until it is barely detectable in the last month of pregnancy Fibrin split products are usually absent in pregnancy unless a complication is present The placenta is rich in antiplasmins, these are not normally released but may represent some degree of protection against postpartum hemorrhage It must be kept in mind that increased levels of certain factors do not automatically result in DIC or thrombosis, some triggering mechanism is needed This system of checks and balances is probably the best protection that the pregnant patient has against the effects of DIC and thromboembolic disease

Changes in the Coagulation System During Normal Labor, Delivery, and Puerperium

Once a normal pregnant woman goes into labor, marked changes occur in the clotting and fibrinolytic systems The partial thromboplastin time shortens, indicating activation of the clotting system while at the same time there is an elevation of factor VII levels, an increase in platelets, and FSP may appear in the blood In the third stage of labor, separation of the placenta is accompanied by a rise in factors V and VII and a fall in the fibrinogen level These factors usually return to normal after 24 hours Fibrinolytic activity begins to return and platelets increase to a peak on the 10th postpartum day, and platelet aggregation is also maximal about this time This platelet peak coincides with the peak time of incidence of postpartum pulmonary embolism

DIAGNOSIS OF DIC

The cardinal clinical manifestation of DIC is abnormal bleeding Hence ICF is probably a better name This is usually in the form of heavy vaginal bleeding If the patient is undergoing an operation, there may be excessive bleeding from the incisional margins other operative areas, and venipuncture sites Hematuria, bleeding from the gums, and ecchymoses are not uncommon, and a full blown shock picture may develop Mental confusion reflects cerebral anoxia, and convulsions may occur

Confirmation by laboratory tests is essential, not only to establish the presence and severity of the disease, but also to monitor its course There is no single laboratory test that is diagnostic of DIC In the full blown syndrome the diagnosis of DIC is easy, as almost all tests will be abnormal, but determination of whether intravascular coagulation or fibrinolysis poses the greatest threat to the patient may be very difficult

A number of laboratory tests and their findings are helpful in the diagnosis and management of DIC (Table 2-2) Some of these are simple to perform and are immediately available to the physician Others require experienced personnel and may take over 24 hours to perform

TABLE 2-2. Laboratory Tests for Disseminated Intravascular Coagulation

Test	Nonpregnant values	Normal pregnancy	Result in DIC
Clotting time	6–12 min	Normal	Normal
Clot retraction	Good	Good	Poor (lyses in 15–60 min)
Fibrinogen	200–400/100 ml	300–500 mg/100 ml	Usually depressed
Thrombin time	12–18 sec	Shortened	Usually prolonged
Prothrombin time	75–125% in 1 hr	Shortened	Usually prolonged
Partial thromboplastin time *or*	40–60 sec	Shortened	Usually prolonged
Activated partial thromboplastin time	25–45 sec	Shortened	Usually prolonged
Factor assays		Normal	V, VIII XIII reduced
Platelets	150 000–400 000/mm³	Normal	Usually decreased
Red cell morphology		Normal	Often abnormal (schistocytes, etc)
Fibrin split products (FDP)		Usually absent	Present
Euglobulin clot lysis		Normal	Usually shortened
Plasminogen		Normal	Usually depressed
Plasma protamine paracoagulation		Fibrin monomer absent	Fibrin monomer present
Ethanol gelation		Fibrin monomer absent	Fibrin monomer present
Protamine sulfate precipitation		Fibrin monomer absent	Fibrin monomer present

When suspicion of DIC is strong, or when clinical evidence of coagulopathy is present, the following battery of readily available tests is suggested 1) quantitative determination of fibrinogen, 2) clotting time, 3) clot lysis time, 4) peripheral smear for platelets and red cell morphology, 5) the Quick prothrombin time determination, 6) partial thromboplastin time determination, 7) thrombin time, and 8) fibrinolytic split products (FSP)

In the absence of good laboratory facilities, the clot observation test and examination of a peripheral smear are valuable For the clot observation test a 5 ml sample of blood is placed in a 15-ml test tube and inverted four or five times The clotting mechanism is abnormal if there is no clot within 6–12 min, or if a clot that forms is not solid and lyses within 1 hour The clot size is abnormal if it occupies less than 45% of the total volume of the blood sample The clot stability is abnormal if, after standing for half an hour, it will not withstand inversion of the test tube several times

The clotting time, as determined in the clot observation test, provides evidence of fibrinogen levels If the clotting time is less than 6 min the fibrinogen level is probably more than 150 mg/100 ml If the clotting time is more than 12 min and the clot is poor, the fibrinogen level is probably 100–150 mg/100 ml If there is no clot in 30 min, the fibrinogen level is probably less than 100 mg/100 ml In this respect however, the quality of the fibrinogen which the patient has available is very important, for the influence of fibrinogen on the clotting process is much greater than that of its first derivative, even though there is little difference in molecular weight Therefore, even though a patient's fibrinogen level may be 200 mg/100 ml, she may still have a significant degree of DIC, and a blood clot that forms promptly may undergo lysis within an hour

A standard blood smear, stained with Wright's stain, can be used to make a rapid diagnosis If fewer than four platelets per high power field are seen this suggests thrombocytopenia This suspicion can easily be confirmed by performing a platelet count Thrombocytopenia is characteristic of intravascular coagulation but is not found in a pure fibrinolytic syndrome Platelets are slow to return to normal even when the process is brought under control In disseminated intravascular coagulation the passage of the red blood cells through fibrin "meshes" changes their shape One can see in a peripheral blood smear "helmet shaped," "tear-shaped" and fragmented red blood cells (schistocytes) In pathologic fibrinolysis alone, the red cell morphology is normal

When good laboratory facilities are available the following three assays are recommended, and ideally should be available at all times These are the prothrombin time (PT), the activated partial thromboplastin time (APTT), and the thrombin time (TT)

The Quick prothrombin time (PT) assay measures the time required for clotting by the extrinsic pathway, and is carried out by adding tissue thromboplastin to plasma In DIC, it is usually prolonged because of the decrease of factors I, II, and X and the anticoagulant properties of fibrinolytic degradation products (FDP) If normal, this finding is not typical of DIC, particularly of the acute type

The activated partial thromboplastin time (APTT) assay is a variant of the PTT (Table 2-2) and like it measures the time required for clotting by the intrinsic pathway This is estimated by adding a surface activator, a platelet substitute, and calcium to plasma It is prolonged in DIC, and is particularly in-

25-45 sec

fluenced by decreases in factors I, II, V and X It is also influenced by the anticoagulant effects of fibrinolytic split products (FSP) If the test is it should be repeated, using a mixture of the patient's plasma and normal plasma As normal plasma replaces all depressed factors, a return to normal would indicate that the high value was due to a deficiency of clotting factors and not to the anticoagulant effects of FDP The usual APTT range is 25–45 sec, but varies with the individual laboratory

The thrombin time (TT) measures the time required for the clotting of plasma after adding extrinsic thrombin It can be influenced only by the levels of fibrinogen and the anticoagulant effects of FDP If the fibrinogen level is normal, a serial TT is useful to detect increased plasma fibrinolytic activity Normal TT is in 12–18 sec

If two or three of these tests are normal, significant DIC can be ruled out. If all are abnormal, in the absence of serious liver impairment, then a presumptive diagnosis of DIC can be made Further testing must be performed to determine whether the abnormality is due to isolated fibrinolysis, ingestion of anticoagulants, or to the effects of massive transfusion with bank blood

Assays of individual clotting factors and evaluation of the fibrinolytic system eventually identify which abnormality is present In DIC, analysis of specific factors reveals diminished levels of factors I, II, V, VII, and XIII, while factors VII, IX, XI, and XII may or may not be depressed, depending upon the severity of the disorder For accuracy of diagnosis, the most helpful are factor I (unaffected by massive transfusion), factor II (unaffected by fibrinolysis), factor V (not affected by vitamin K deficiency or oral anticoagulants) and factor VIII (not depressed in liver disease)

Evaluation of the fibrinolytic system is important since the degree of activation of fibrinolysis can affect the course of the disease The clotting time of whole blood or plasma is relatively insensitive as only in cases of very severe systemic fibrinolysis will the clot lyse within a short time A more useful test is the euglobulin lysis time (ELT), which measures fibrinolytic activation Extreme shortening of the ELT reflects excessive systemic fibrinolytic activity provided sufficient fibrinogen is present In consumption coagulopathy the ELT can be normal, shortened, or prolonged

One of the most consistent findings in DIC is the appearance of FSP These can be detected in serum, and their reaction with antifibrinogen serum in the presence of latex particles produces agglutination Semiquantitation may be achieved by using serial dilutions of serum Prolongation of thrombin time, in the absence of heparin treatment, and with normal fibrinogen levels, usually reflects significant circulating concentrations of FDP

The ethanol gelatin test, the protamine sulfate precipitation test, and the *staphylococcus clumping test can be used to detect soluble* fibrin monomer complexes This is important because the formation of fibrin monomer is probably a first step in the process of intravascular coagulation These fibrin monomers may be complexed with fibrinogen and remain soluble in the blood until removed by the reticuloendothelial system, or they may be deposited in the blood vessels by further activation of the thrombotic mechanism

In 1970, Seaman described the plasma protamine paracoagulation (PPP) test, and in 1975, Phillips reported on its use in 500 obstetric patients The value of this test lies in the fact that fibrin monomer is readily detected by this simple rapid test. In Phillips' series, the incidence of positive fibrin monomer was less than 1% in 139 blood samples drawn during normal pregnancy In

abruptio placentae, 84% of samples were positive, but in the presence of other types of antepartum hemorrhage they were absent The percentage of positive tests with intrauterine dead fetus syndrome was 24%, with eclampto genic toxemia it was 19%, and with sepsis it was 18% In blood samples drawn from patients 3–48 hours after elective saline abortion, 33% were positive, as compared with 3% following prostaglandin administration The results correlated well with the clinical condition of the patients, and holds considerable promise because it is simple, and a result can usually be obtained within 30 min

MANAGEMENT

The amount of bleeding provides a guide to the management of DIC (Table 2-3) However, this approach must be modified according to the rest of the clinical picture and the coagulation profile at the time when the patient is seen In a patient with abruptio placentae, prompt evacuation of the uterus is more important than heparin, unless the coagulopathy is prolonged On the other hand, in more chronic conditions such as intrauterine dead fetus syndrome (Fig 2–5), coagulation should be restored to normal by the administration of clotting factors and therapeutic doses of heparin prior to delivery

DIC With Obvious Bleeding

When obvious bleeding accompanies DIC (as in abruptio placentae), in general the following steps should be taken if hemorrhage is brisk and the main problem appears to be a coagulation defect

1. Monitor vital signs and central venous pressure (CVP)
2. Give oxygen by face mask
3. Replace blood loss promptly, with fresh blood if possible
4. Give fresh frozen plasma to supply multiple clotting factors and volume replacement (Each liter of fresh frozen plasma supplies factors V and VII as well as 2 g fibrinogen, with less risk of hepatitis than when fibrinogen is used)
5. Give cryoprecipitate This supplies fibrinogen and factor VIII, and 5–25 units may be required Fibrinogen (2–6 g) is given only if other measures fail or are not available
6. Give heparin only if factor replacement is not adequate to control bleeding The initial infusion is begun at 1000 units/hour
7. Epsilon aminocaproic acid (EACA) should be used only if marked fibrinolysis is evident and heparin is used concomitantly The initial dose is 4 g intravenously, and then 1 g every 4 hours is given as required
8. Remove the source of thromboplastin, so that no further therapy is required (This may be the first step in some patients)

DIC Without Hemorrhage

If there is no hemorrhage with DIC (as in the dead fetus syndrome), give heparin, 1000 units/hour, up to 600 units per kilogram of body weight in 24 hours, by intravenous infusion until clotting factors are in the normal range The source of thromboplastin is then removed if possible

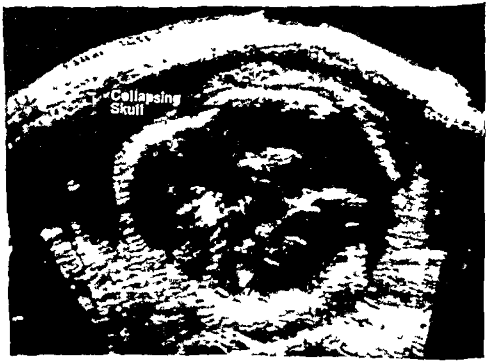
Further detailed management of particular shock states involving DIC are considered in Chapter 3, Shock.

TABLE 2 3 Treatment of Bleeding in Intravascular Coagulation Fibrinolysis Syndrome

Treatment	Bleeding state			
	Mild	Moderate	Severe	Life-threatening
Treat pr mary d sease process	+	+	+	+
Replace def cient clott ng factors		+	+	+
Inh b t clott ng process with hepar n			+	+
Inhib t f brinolysis with EACA				+

(Owen CA Jr Bowie EJW The Intravascular Coagulation Fibr nolys s Syndromes i Obstetrics and Gynecology The Upjohn Company Kalamazoo MI 1976)

FIG 2 5 Sonogram Intrauterine fetal death Halo sign with collapsing bo structure (Courtesy of Dr R E Woods)



IDIOPATHIC THROMBOCYTOPENIC PURPURA

Idiopathic thrombocytopenic purpura (ITP) is an autoimmune disorder in which antiplatelet antibodies shorten the life span of the platelets

The diagnosis is made when a patient has 1) thrombocytopenia 2) vascular fragility as measured by a positive Rumple Leede test and increased bleeding time and 3) a marrow smear showing a normal or increased *megakaryocyte count with many young forms*

The importance of ITP in pregnancy lies in the danger that it holds for oth mother and baby

The condition may cause severe bleeding in the mother at cesarean section r from cervical or vaginal lacerations at the time of delivery It may also ause postpartum uterine hemorrhage or vulvovaginal hematoma formation Jeonatal thrombocytopenia occurs in approximately 50% of cases, and the erinatal mortality is about 20%

MANAGEMENT

Steps in management are as follows

1. Corticosteroids are given if the diagnosis is made during pregnancy
2. Platelet transfusions are given to maintain the platelet count at 100,000/cu mm prior to induction or onset of labor, during labor, at cesarean section, or if severe hemorrhage occurs
3. Splenectomy is best postponed until after delivery, but may be required if steroids do not effect a satisfactory remission
4. In a severe case, elective cesarean section should be considered, to minimize trauma to the fetus

VON WILLEBRAND'S DISEASE

Although rare, this disorder is probably the most common congenital clotting defect in American women of childbearing age It is an incompletely dominant autosomal trait

The diagnosis is suspected in a patient with a history of a familial bleeding tendency, previous bleeding episodes, a prolonged bleeding time, a mild to moderate factor VIII deficiency, and a tendency to mucocutaneous hemorrhage The most useful diagnostic tests are presented in Table 2-4

The importance of this condition in pregnancy is due to the increased maternal morbidity, Problems are usually associated with operative delivery Perinatal mortality is not increased

MANAGEMENT

The management of bleeding episodes consists of replacement of factor VIII In an emergency, factor VIII concentrate, or 4 units of cryoprecipitate are given intravenously immediately This is followed by 2 units of cryoprecipitate every 3 hours

If factor VIII is less than 30% of normal and operative delivery or episiotomy is anticipated, cryoprecipitate should be given If time permits, adequate levels of factor VIII can be achieved by giving 1 unit of fresh frozen plasma per day, or up to 1 bag of cryoprecipitate per 10 kg of body weight per day

CIRCULATING ANTICOAGULANT ANTIBODY SYNDROME

In rare instances, patients develop an antibody to factor VIII, with the production of pseudohemophilia This antibody may pass the placental "barrier" and affect the fetus

TABLE 2-4 Diagnostic Tests for Von Willebrand's Disease

Test	Result
Bleeding time	Prolonged
Clotting time	Normal
Partial thromboplastin time	Prolonged
Platelet count	Normal
Platelet adhesiveness	Reduced
Factor VIII level	Reduced
Tourniquet test	Positive

The diagnosis should be suspected when the PTT is prolonged and factor VIII is depleted

The management is as for Von Willebrand s disease

FACTOR XI DEFICIENCY

This defect is inherited as an incompletely recessive autosomal trait It may be major (level below 20% of normal) in a patient homozygous for the defect or minor (level 30–60% of normal) in a patient who is heterozygous

The importance of the defect lies mainly in the fact that excessive bleeding may occur with surgery when factor XI is less than 50%

The diagnosis is suspected when the partial thromboplastin time is prolonged and is confirmed by specific tests for factor XI

The management consists of maintaining factor XI levels above 50% of normal This is achieved using an infusion of stored plasma in a dosage of 10 ml/kg/day

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Chapter 3

Shock

Denis Cavanagh

A little fire is quickly trodden out
Which being suffered rivers cannot quench
—William Shakespeare (1564–1616)
King Henry VI Part III Act IV Scene 8

Every obstetrician gynecologist must be familiar with the early recognition and the correct management of shock Failure to do so results in the death of many women every year all over the world In addition to the toll in avoidable maternal deaths shock also accounts for a large number of deaths among gynecologic patients Moreover, some of the most serious complications of pregnancy, such as Sheehan s syndrome, bilateral renal cortical necrosis and acute tubular necrosis, are caused by shock For the early diagnosis and the proper management of the patient 'in shock, an understanding of the basic mechanisms of the syndrome is essential Moreover, these mechanisms must be related to a knowledge of the hemodynamic changes of normal pregnancy

Shock may also occur with the use of some of the antibiotics used to combat life-threatening infections during pregnancy which have not been approved for use in pregnancy by the Food and Drug Administration (USA), and so the benefit to the mother must be balanced against possible damage to the fetus

Profound alterations in the circulatory system occur during pregnancy Some of these changes predispose to the development of shock while others apparently protect against it

Most of the hemodynamic changes of pregnancy appear to be the result of 1) the presence of a modified arteriovenous shunt at the placental site and 2) obstruction to the venous return to the heart by the gravid uterus The systolic blood pressure is generally reduced and the diastolic pressure almost invariably so, in normal pregnancy The pulse rate is elevated by 12–20 beats per minute Blood volume is maximal from 32 to 36 weeks and cardiac output from 25 to 27 weeks The work load on the heart is increased in spite of the reduction of peripheral resistance Peripheral blood flow is increased and in addition precapillary arterioles and the capillaries show in

creased vasomotor activity Increased capillary fragility probably occurs dur the third trimester and certainly does during labor There are also changes in t coagulation mechanism that predispose to the development of disseminated int vascular coagulation and a hemorrhagic diathesis (see Ch 2, Clotting Disord in Pregnancy)

The term shock has been applied for over 200 years to a variety of cor tions in which the original traumatic insult seemed disproportionately sn when compared to the ultimate clinical picture The word was apparer first used in the English language in 1743 in a translation of Henri Francoi Dran's second French edition of *A Treatise of Reflections Drawn from i perience with Gun Shot Wounds* The term was used to convey the seque of a blow, followed by progressive deterioration, loss of consciousness, death Simeone quotes a definition of shock given by Gross in 1872, who scribed the condition as "a rude unhinging of machinery of life" Anot colorful definition is that of John Collins Warren who described the co tion as "a momentary pause in the act of death" These definitions sup the current concept that shock is not an entity in itself but rather a bo response to a life threatening situation that, if not corrected, results in per nent damage to vital organ systems

According to Wiggers, in his monumental monograph on shock publis in 1950, laboratory research into shock began at the turn of the 20th tury with the work of George W Crile, the prominent Cleveland surg Crile was not only the first person to search for physiological explanations the syndrome, but he also called attention to the importance of changes in peripheral circulation Henderson in 1910, described the relationship tween venous return, cardiac output and arterial blood pressure as follow

> Because of the diminished venous supply the heart is not adequately diste and filled during diastole Hence the picture of a "failing heart revealed by pulse For the same reason arterial pressure ultimately sinks in spite of int activity (not because of failure) in the vasomotor nervous system and in of an extreme constriction (not because of relaxation) of the arterioles

DEFINITION

Shock is a condition in which there is a disparity between the circul blood volume and the capacity of the vascular bed This disparity re in hypotension and more importantly in reduced tissue perfusion of organs producing cellular hypoxia If uncorrected, these events will lead progressive failure of cellular metabolism and eventually to vital o *damage and death Note that in this definition the* emphasis is placed on curtailment of tissue perfusion, rather than on the increase in heart rate the decrease in blood pressure, which usually accompany it The reduc in tissue perfusion results in deficient transfer of oxygen and other nutri to the cells and inadequate removal of carbon dioxide and waste prod of cellular metabolism In early shock, anaerobic metabolism compens for oxygen deficiency If adequate tissue perfusion is not restored prom however, the continued anaerobic metabolism of glucose leads to accum tion of lactic and pyruvic acid with the development of metabolic acid

Progressive cellular hypoxia results in enzymatic and metabolic dysfunction, lysosomal disruption with liberation of hydrolases and ultimately cellular destruction

INCIDENCE

The exact incidence of shock in obstetric and gynecologic patients is unknown In some complications, such as ruptured uterus, it is almost always present, whereas according to Douglas and Bechman, it complicates only 2 5–6 4% of cases of infected abortion When shock is present it complicates an underlying disease process, causing great physiologic disturbance and increasing the threat to the life of the patient In the pregnant woman the living fetus is also at risk, for maintenance of adequate placental perfusion is essential to fetal well-being

CLASSIFICATION

One of the problems in evaluating the effectiveness of the management of shock is that the results vary according to the primary cause In addition, they vary according to the stage of shock at which a patient is first seen Shock may arise from any one of a number of causes Hypovolemia is the most common cause Shock due to infection, cardiac failure, hypersensitivity (anaphylactic or allergic), neurogenic disturbances, or blood flow impedance is also seen

There are many classifications of shock The following classification, especially applicable to shock in women, is based on the initiating hemodynamic event and includes complications seen in obstetric and gynecologic patients *

1. Hypovolemic shock
   - A. Hemorrhagic shock Associated with postpartum or postabortal hemorrhage, ectopic pregnancy, placenta previa, abruptio placentae, rupture of the uterus, dysfunctional uterine bleeding, benign and malignant uterine neoplasms, rupture of ovarian neoplasms, and obstetric and gynecologic surgery
   - B. Fluid loss shock Associated with excessive vomiting, diarrhea, diuresis, or too rapid removal of ascitic fluid in patients with hepatic cirrhosis
   - C. *Supine hypotensive syndrome* Associated with *compression of inferior* vena cava by pregnant uterus
   - D. Shock associated with disseminated intravascular coagulation Intrauterine dead fetus syndrome and amniotic fluid infusion
2. Septic shock (*endotoxic shock*) Associated typically with infected abortion, chorioamnionitis, pyelonephritis, and postpartum endometritis, may be hypovolemic, has cardiogenic component
3. Cardiogenic shock
   - A. Failure of left ventricular ejection
     - 1) Cardiac arrest (asystole or ventricular fibrillation)
     - 2) Myocardial infarction

* Adapted from Cavanagh D, Comas MR In Romney S Gray MJ, Little B et al (eds) Gynecology and Obstetrics The Health Care of Women New York McGraw Hill, 1975

B. Failure of left ventricular filling
   1) Cardiac tamponade Associated with coagulation defects
   2) Pulmonary embolism Associated with infusion of air or fat, or thrombophlebitis associated with pregnancy, use of hormones, ... tensive pelvic surgery, or sickle-cell disease
4. Neurogenic shock
   A. Chemical injury Associated with aspiration of gastrointestinal conten
   B. Drug induced Associated with spinal anesthesia
   C. Inversion of uterus with vasomotor collapse
   D. Electrolyte imbalance Associated with hyponatremia from any cause

The conditions cited in the list may operate independently or in combinatio When a pregnant woman is in shock, the fetus is also in shock

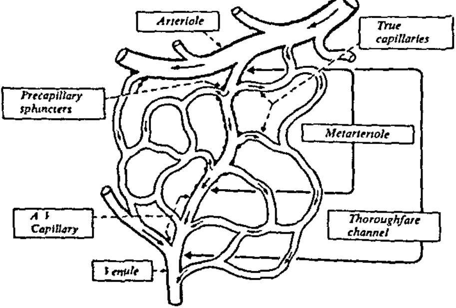
HYPOVOLEMIC SHOCK

As already stated shock is caused by a critical reduction in the perfusi of tissues by blood A review of how the microcirculation normally perfuse the tissues, and then how this process is affected by shock is therefore im portant

A microcirculatory unit (Fig 3-1) consists of blood vessels arrange serially and in parallel including arterioles metarterioles, precapillary sphinc ters, capillaries, arteriovenous anastomoses, venules, and collecting venules

Functionally, the components of the microcirculation may be classified as re sistance vessels, exchange vessels, shunt vessels, and capacitance vessels

FIG 3 1 Diagram of microcirculatory unit (Selkurt E Physiology, 3rd ed Boston Little, Brown 1971 after Chambers R and Zweifach BW)



RESISTANCE VESSELS The precapillary resistance elements include small arteries, arterioles, and precapillary sphincters The first two elements determine the extent of total blood flow to the tissues while the sphincters, by adjusting the number of capillaries open, determine the distribution of capillary blood flow, the velocity of capillary flow, capillary surface area available, and the mean extravascular diffusion distance Postcapillary resistance vessels include the muscular venules and small veins Their strategic position, immediately postcapillary, enables them to influence capillary pressure markedly It is the ratio of precapillary to postcapillary resistance that determines the capillary hydrostatic filtration pressure

EXCHANGE VESSELS These are the capillaries and venules The exchanging between the vascular and extravascular compartments is carried out through their walls in a variable manner In any given tissue the venous end of the exchange vasculature is more permeable to water and solutes than is the arterial end

SHUNT VESSELS Included among these vessels are arteriovenous anastomoses, preferential channels, and even capillaries when they receive blood flow greatly in excess of their exchange capacity Flow through these vessels bypasses the effective exchange circulation of tissue, and therefore is considered nonnutritional

CAPACITANCE VESSELS Because of their great distensibility, the veins are the major capacitance or storage elements of the circulation The venous component of the microcirculatory system is estimated to contain about 70% of the total body blood volume

VASOMOTION Direct examination of the normal microcirculation reveals that blood flow is very intermittent Flow in any single capillary depends primarily upon the state of the precapillary resistance vessels These vessels exhibit vasomotion or alternate phases of contraction and relaxation With augmented vasomotion, the frequency of contraction and relaxation increases, and the contraction phase becomes more prominent, thereby diminishing capillary blood flow Conversely, a reduction of vasomotion involves a reduction in the frequency of the cycle with the dilator phase predominating, and capillary blood flow increases

Vasomotion is controlled by local chemical, myogenic, and neurogenic influences acting upon the vascular musculature Arteriolar vasomotion is probably influenced more by central nervous impulses which travel through the sympathetic outflow to the vessels, than by local humoral influences At the metarteriole and precapillary sphincter level, neurogenic influences appear to act mainly by modifying the reaction of the vascular smooth muscle to other influences Here, vasomotion depends to a large extent on the level of tissue activity When tissue metabolism increases or when a degree of anaerobic metabolism develops, as in shock, there is an increased accumulation of metabolic waste products These depress the smooth muscle elements of the peripheral vasculature, thus reducing vasomotion and increasing capillary blood flow Conversely, when metabolic activity is minimal, tissue metabolites

are at a lower concentration and vasomotion is maintained at a relatively high frequency, thus decreasing capillary blood flow

The postcapillary resistance elements, the muscular venules, and small veins are mainly responsive to neurogenic impulses Increased sympathetic activity increases their smooth muscle contraction (venomotor tone) and reduces their capacitance Constriction of capacitance vessels occurs at a neural discharge rate even lower than the rate at which the resistance vessels respond. When necessary, this constriction decreases peripheral vascular volume and thereby acts to increase the filling pressure of the heart Loss of this tone can result in serious hypotension by reducing cardiac filling

How is the microcirculation affected by the shock state? Consider first the circulatory changes which take place in hemorrhage shock

HEMORRHAGIC SHOCK

In general, the economy of the body is normally maintained by a process of negative feedback, that is, the response to a noxious stimulus tends to cancel or correct the drift from the norm After severe hemorrhage the patient goes into a state of primary or "reversible" shock The resulting hypotension stimulates stretch receptors monitoring blood pressure in the carotid sinus and aortic arch These supply information to the vasomotor center in the medulla via the ninth and tenth cranial nerves The vasomotor center provides a response designed to restore the blood pressure to its former value. The efferent limb of this reflex is mediated through the sympathetic nervous system This produces an increase in the rate and force of cardiac contraction, which improves the effective cardiac output This sympathetic discharge also constricts the peripheral arterioles and increases the tone of the venules and small veins Blood flow through nonvital organs is diminished, and the large venous reservoir is emptied into the central circulation This also causes a fall in the hydrostatic pressure across the tissue capillaries, and fluid will then move from the extravascular compartment into the circulation Human volunteers who bleed 15% of their blood volume over a period of 30 min show an autoinfusion rate of approximately 2 ml/min for the first 2 hours and an average of 50/ml hour for the next 6–10 hours In patients with severe hemorrhage the plasma refill rate is even more rapid

Control of blood flow through the brain and the heart is mediated almost entirely by local factors, therefore, these organs do not participate in the "sympathetic squeeze" The three defense mechanisms brought into play are 1) selective organ ischemia, 2) immediate autotransfusion from increased venomotor tone, and 3) delayed autotransfusion from transcapillary refilling These mechanisms correct hypovolemia, improve cardiac output and sustain blood pressure and therefore the perfusion of vital organs At this stage, transfusion and control of hemorrhage are usually effective in restoring the normal circulatory balance

Other responses help stabilize the plasma volume Increased release of antidiuretic hormone by the pituitary and of aldosterone by the adrenal cortex, result in the conservation of sodium and water by the kidneys Most patients in hypovolemic shock exhibit acute thirst If oral intake of fluids were desirable this would probably restore plasma volume All the responses mentioned are examples of negative feedback mechanisms functioning to correct the drift from the normal state

When early and adequate measures are not taken to correct the disparity etween the circulating blood volume and the capacity of the vascular bed, :condary or "irreversible" shock will develop Here, the initial compensatory iechanisms fail, and "positive feedback" cycles are established, in which the iitiating stimulus is increased by the response The sequence of events is as ollows The available blood volume continues to fall Excretion of metabolites ; deficient from areas undergoing severe vasoconstriction The accumulation f these metabolites eventually constitutes a powerful metabolic stimulus *or vasodilation of the metarteriole and the precapillary sphincter in spite of* ie persistence of increased sympathetic tone Postcapillary resistance ele-ients, however, apparently *continue in a state of constriction long after the pre-*apillary sphincters have begun to dilate, thereby causing the blood to be ooled in the capillary bed This increases the capillary hydrostatic pressure nd decreases or even reverses the fluid shift from the extravascular to the ascular space Venous return to the heart is further decreased, with a con-equent drop in cardiac output and blood pressure As the diastolic pressure alls, coronary artery blood flow is reduced producing myocardial hypoxia nd eventually cardiac decompensation This cardiac injury can be over-vhelming and self-perpetuating

In the final states of hemorrhagic shock, the postcapillary venules become inresponsive to sympathetic stimulation, and the microcirculation is then ubjected *to the unopposed effect of endogenous vasodilator substances* The :apillary and venule spaces open up This results in progressive pooling of ılood and stagnant hypoxia This is the stage of secondary or irreversible hock At this time, no amount of blood replacement, ventilation, drugs, or urgical manipulation will reverse the course, and the patient will soon die of cardiorespiratory failure

The previously described effects of hemorrhagic hypovolemic shock on he microcirculation are a classic example of the low output shock syndrome t is possible, however, to have shock without hypovolemia and even with a 'hyperdynamic" circulatory state This can be seen in certain patients with eptic shock of endotoxin type

Pathophysiology

The central event in hemorrhagic shock is loss of blood from the vascular space with diminution of circulating blood volume *In its compensated,* reversible phase, hemorrhagic shock is featured by elevation of circulating catecholamines with widespread arteriolar and venous *constriction* Liberation of pressor substances helps sustain this vasoconstriction, and renal blood flow is redistributed within the kidney to protect *the medulla* Splanchnic, uterine, renal muscular, and cutaneous flows decrease, and the brain and heart are benefited by preferential channeling of blood *There is also an influx of* extravascular fluid into the vascular compartment, the net effect being the ac-commodation of the available circulating volume to the vascular space Venous return and cardiac output are thereby maintained Tachycardia also helps maintain cardiac output In this phase of shock, *administration of intravenous* fluids and electrolytes achieves homeostasis readily, provided that hemorrhage is controlled

With continued blood loss, hemorrhagic shock enters the primary stage of early decompensation Peripheral vasoconstriction, *influx of extravascular*

fluid into the vascular space, and tachycardia no longer preserve circulating volume because of continued fall in the circulating blood • With tissue hypoxia, increased capillary dilatation occurs, possibly the of local histamine liberation This dilatation further increases the between the available blood volume and the capacity of the vascular bed that venous return and cardiac output are further reduced The process intensified by sequestration of fluid through capillary walls damaged by poxia

If allowed to continue, the process enters the secondary stage of late d compensation, in which arteriolar and capillary tone is lost, with vast sion of the capillary bed and therefore of the vascular compartment in eral Marked metabolic acidosis occurs at times associated with d intravascular coagulation (DIC) and stimulation of the fibrinolytic •, The transition from this phase into the stage of irreversible hemorrhagic is signaled by evidence of organ death in the form of hepatic, renal, cardic pulmonary or central nervous system failure

Clinical Picture

From a clinical point of view hemorrhagic shock may be divided as fol lows

1. Primary shock (reversible)
   A. Early (warm) or compensated
   B. Late (cold) or decompensated
2. Secondary (irreversible)

In its early phases, hemorrhagic shock presents a relatively normal bloo pressure, tachycardia, and diaphoresis The patient appears restless an anxious (Table 3-1) This compensated phase is easily managed by volum replacement If untreated, it is followed by the hypotensive phase In its earl stages, this decompensated phase also responds readily to adequate volum replacement Later in the evolution of this process, however, a much le satisfactory response is elicited, even though treatment is intensified, and th patient may enter the secondary or irreversible stage Hemorrhagic shock characteristically reversible until very late in its evolution For this reaso vigorous therapy should always be initiated when the diagnosis is made, eve in an apparently exsanguinated patient

Management

GENERAL PLAN OF ACTION Weil and Shubin (1976) have emphasized th importance of placing a priority on the sequence of therapeutic and dia nostic maneuvers (Table 3–2), the order of priority giving the mnemon "VIP-PS" (Ventilation Infusion Pump Pharmacologic Surgical treatment This approach is much more valuable in the bedside management of patien in shock than are such time-honored routines as the prolonged use of th head down position, which only increases the work of breathing in an alread hypoxic patient

Ventilation is essential because the most common cause of death in shoc in inadequate respiratory exchange Measurement of the pH, the oxyge saturation (Po_2), and the carbon dioxide pressure (Pco_2) of arterial blood important When the Po_2 in arterial blood is less than 70 torr (93% satur

ABLE 3-1 The Clinical Picture in Hemorrhagic Shock and Expected Response to olume Replacement

	Primary shock (early)	Primary shock (late)	Secondary shock
Mental state	Alert and anxious	Confused	Coma
General appearance	Normal and warm	Pale and cold	Cyanotic and cold
Blood pressure	Slightly hypotensive	Moderately hypotensive	Markedly hypotensive
Respiratory system	Slight tachypnea	Tachypnea	Tachypnea and cyanosis
Urinary output	30–60 ml/hr	<30 ml/hour	Anuria
Effect of volume challenge on			
Blood pressure	Increased	Slightly increased	No response
Urinary output	Increased	Slightly increased	No response

TABLE 3-2. Sequence of Therapeutic Diagnostic Maneuvers in Shock

Priority	Mnemonic	Therapy	Purpose
1	V	Ventilate	Adequate pulmonary CO_2 and O_2 exchange
2	I	Infuse	Blood fluid electrolyte balance
3	P	Pump	Restoration of cardiac competence
4	P	Pharmacologic	Use of vasoactive agents to improve perfusion
5	S	Specific surgical	Medical and surgical management of primary causes

(Weil MH Shubin H [eds] Critical Care Medicine Hagerstown MD Harper & Row 1976)

tion), and if the P_{CO_2} is more than 46 torr, and the pH is less than 7 35, respiratory acidosis from inadequate exchange is present The arterial P_{O_2} must be returned to normal as soon as possible to reduce tissue hypoxia

Infusion of an adequate volume of blood, colloids (5% serum albumin), or crystalloids is essential to combat shock The central venous pressure (CVP), the pulmonary artery wedge pressure, the blood volume estimation, and the urinary output are useful guides to the fluid intake requirements Cardiac competence may be assessed in a patient with a borderline CVP (12–16 cm H_2O) by infusing 500 ml fluid at a rate of 20 ml/min If the CVP does not rise more than 5 cm H_2O and returns to within 2 cm of the initial level, the myocardium is competent The *pump* must be effective, so cardiac competence must be maintained or restored, so as to achieve an adequate circulating blood volume

The *Pharmacologic* and *Surgical* treatment of shock are usually only begun after the VIP priorities have been fulfilled

DETAILED MANAGEMENT The essentials of management in hemorrhagic shock are to stop bleeding and replace blood After the diagnosis of hemorrhagic

shock has been made, hemostasis is essential However, this may be only by major surgery Unless definite evidence of intra abdominal ' is present, surgery should be deferred until measures for control of have been initiated When indicated, such measures as oxytocin tion for uterine atony, or repair of a cervical laceration should be without delay

Replacement of intravascular fluid volume is essential, and ideally should be replaced with blood This should always be done through a bore needle (18 gauge), or indwelling polyethylene catheter (16-gaug to allow rapid replacement Early in the development of hemorrhagic sho administration of intravenous fluids and electrolytes may be used Five p cent glucose in normal saline solution is of value, with sodium added as needed to combat acidosis Ringer s lactate solution is less because in shock lactate is already being produced in large amounts anaerobic metabolism The conversion to bicarbonate that occurs during aerobic metabolism is obviously inefficient in this situation

With more severe degrees of hemorrhagic shock adequate replacement properly typed and crossmatched blood becomes essential In the face of hemorrhage, the blood should be as fresh as possible Sufficient blood sho be given to replace the estimated blood loss or until all clinical evidence shock has subsided When blood is not available, serum albumin dextr or 3% saline may be used

In the obstetric patient because of her great ability to compensate for bl loss, the arterial blood pressure is a poor guide to the management of hem rhage This is particularly so in the patient with abruptio placentae, in wh central venous pressure monitoring is a much more accurate guide to repl ment As O Driscoll (1966) has stated

> Our experience of central venous pressure control in 70 severe cases of abru placentae has been that when the uterus was tense and tender the average tr fusion required to restore the venous pressure to normal was 1 5 liters and w there was also vasoconstriction as indicated by skin pallor the average w liters The average transfusion required to restore the venous pressure when arterial pressure was reduced was a massive 5 liters

Early and adequate replacement is especially difficult to achieve in abrup because of the tendency to underestimate blood loss

Other therapeutic measures should include oxygen administration by n catheter at a flow of 4–5 liters per minute and the use of the modi Trendelenburg position (feet elevated head down) during the first few m utes of treatment The use of vasopressors should be kept to a minimum si peripheral vasoconstriction already exists Therapy should be monitored repeated readings of central venous pressure initially every hour less quently as improvement occurs Hourly urinary output should be recor together with frequent observations of pulse respirations and blood press Blood volume estimations are useful in early shock but are quite inaccu in late shock Hemoglobin and hematocrit determinations are of little v in ascertaining the magnitude of acute blood loss but a base line determi tion followed by daily measurements provides some guidance as to adequacy of replacement therapy The best immediate guide to ther is the clinical appearance of the patient Tachycardia and diaphoresis

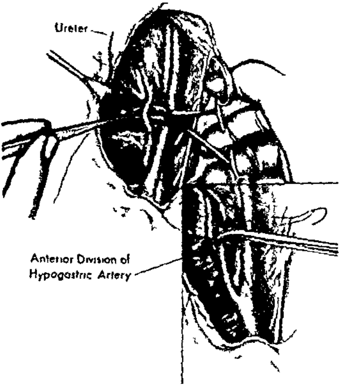
especially useful signs Blood pressure alterations frequently appear only when the process is more advanced

When therapeutic measures to combat shock have been initiated, surgical efforts to attain hemostasis may be undertaken The magnitude of the surgical procedure depends of course upon the underlying condition Although it is usually preferable to defer surgery until shock has been combatted it is sometimes impractical to await response to medical measures, as in severe hemorrhage from rupture of the gravid uterus In such cases control of hemorrhage must be achieved by laparotomy When a blood filled abdomen is opened manual compression of the aorta against the vertebral column will often allow better visualization of the bleeding site, after the removal of clots

Identifiable bleeding points can be controlled with proper suturing Rupture of the gravid uterus usually calls for total abdominal hysterectomy In rare cases, if the defect is small, repair may be attempted However, there is always the hazard of another rupture with a subsequent pregnancy

Ligation of the hypogastric arteries (Fig 3 2) is a useful technique in

FIG 3 2 Ligation of right hypogastric artery Peritoneum is opened lateral to bifurcation of common iliac artery, and ureter is reflected medially with peritoneal flap *There is much variation in length and point of division* of hypogastric artery into anterior and posterior branches If possible, anterior branch should be selected for ligation and silk suture placed around it **Inset.** *Anterior division of hypogastric artery ligated and excised*, and transfixion ligature placed distal to initial silk tie (TeLinde RW, Mattingly RF Operative Gynecology, 4th ed Philadelphia, Lippincott, 1970)



certain situations in which hysterectomy is impossible or undesirable Th peritoneum should be incised over the external iliac artery and the incisio carried proximally lateral to the ovarian vessels The ovarian vessels are ligat bilaterally prior to ligation of the hypogastric arteries The ureter should al ways be identified prior to the performance of bilateral hypogastric ligation It is usually easily identified as it crosses the common iliac artery The bi furcation of the iliac arteries is isolated The internal iliac artery is identified and its anterior division is doubly ligated with 1 0 silk suture close to its origin Few adverse effects result from this procedure Usually both hypo gastric arteries should be tied, but they should not be transected

Smith and Wyatt (1977) have described a method for control of vaginal bleeding by embolization of the hypogastric arteries, but further experience with this technique will be necessary before its value is established

FLUID LOSS SHOCK

This may be seen in association with excessive vomiting, diarrhea, or exces sive diuresis The diagnosis is obvious, and the management is by adequa replacement with appropriate fluids using serum electrolyte values as guide

SUPINE HYPOTENSIVE SYNDROME

Although not the result of hemorrhage or infection this condition ma cause alarm in the antepartum period The hypotension in these wom is believed to be due to reduced venous return to the right auricle fro the pressure of the gravid uterus on the inferior vena casa Typically, t patient has been asked to lie on her back in bed or on an examining table, a while her blood pressure is being taken an acute hypotensive episode occu The situation is quite alarming to attendants unfamiliar with the supine hyp tensive syndrome, but the blood pressure returns promptly to the norm range when the patient is turned on her side Women should be discoura from lying on the back in late pregnancy, because this position may cau hypotension in the mother and distress to the fetus

Since the elucidation of this syndrome, more consideration is being giv to vaginal delivery in the left lateral position, and even the performance cesarean section with the operating table tilted 10° laterally to the left

SHOCK DUE TO INTRAVASCULAR COAGULATION AND FIBRINOLYSIS

Intrauterine Dead Fetus Syndrome

Most fetuses are delivered spontaneously soon after intrauterine death Trico and Kohl (1957) reported that if the patient is allowed to go into labor spo taneously, 75% of patients will be delivered by the end of the second we after fetal death, and over 90% will do so by the end of the third week

If the dead fetus is retained in utero for longer than 5 weeks, the moth may develop DIC. This is the result of passage of small but repeated infusio of thromboplastic material from the degenerating placenta and dead fe

into the maternal circulation It is usually a slow process, and the defibrination syndrome develops over a period of days to weeks According to Pritchard (1959), once the fetus is retained longer than 5 weeks the likelihood of significant DIC will develop is about one in four

MANAGEMENT Until recently, the management of these patients has been conservative, with the obstetrician usually awaiting the onset of spontaneous labor, and doing weekly coagulation profiles or fibrinogen determinations after the fourth week Most obstetricians now choose a policy of delivery if labor has not ensued by this time This reduces the danger from DIC and certainly reduces the emotional stress on the mother The safest method of inducing labor is to give an intravenous infusion of oxytocin solution in very gradually increasing concentrations until labor is established If an infusion pump is available, it should be used, because excessive contractions may induce amniotic fluid embolism The use of intraamniotic hypertonic saline to induce labor is not recommended because this technique itself may produce DIC

If the patient does develop a coagulation defect, heparin (600 units per kilogram of body weight per 24 hours) should be given by intravenous infusion, with the total dosage depending on response This usually restores fibrinogen and other coagulation factors to normal levels When the coagulation profile has returned to normal, labor can then be induced and delivery safely accomplished Fibrinogen replacement therapy, preferably with cryoprecipitate, should be limited to patients who have very low fibrinogen levels and heavy bleeding The use of fresh blood and heparin is generally to be preferred because fibrinogen administration carries a considerable risk of serum hepatitis, and may perpetuate intravascular coagulation by adding grist to the mill Epsilon-aminocaproic acid (EACA) should be used only when fibrinolysis is marked, and, even then, heparin should be given with it (see Ch 2, Clotting Disorders in Pregnancy)

Amniotic Fluid Embolism (Infusion)

In 1926, Meyer first reported a case of amniotic fluid embolism that produced sudden death in a 21-year-old multipara Lushbaugh and Steiner (1942) solidly delineated the condition with a small series of well documented cases The mortality is very high Approximately 50% of patients die at the time of embolization, and over half the survivors die subsequently Significant amniotic fluid embolism is rare Albrechtsen (1964) estimates that it occurs approximately once in 37,000 deliveries, but it may be more common than this and may account for up to 10% of maternal deaths Predisposing factors include multiparity, hypertonic uterine contractions, oxytocin induction or stimulation of labor, traumatic delivery, meconium staining on the amniotic fluid, large babies, and intrauterine fetal death Although usually seen at term, a case has been reported as early as the 20th week of pregnancy

DIAGNOSIS The diagnosis is often difficult, and the situation has been further complicated because of a tendency to blame amniotic fluid infusion for all cases of unexplained maternal death or unexplained "obstetric shock." The condition is often heralded by a shaking chill, sweating, anxiety, coughing, vomiting and convulsions Courtney (1974) points out that the five cardinal signs are

1. Respiratory distress
2 Cyanosis
3. Cardiovascular collapse
4 Hemorrhage
5. Coma

Pulmonary edema with pink, frothy sputum and absence of chest pain amniotic fluid embolism rather than pulmonary infarction If the pat survives the initial shocklike episode, bleeding (secondary to coagulopathy) and uterine atony may occur

How amniotic fluid triggers clotting is not clear, because it has little plastic activity (it may be the 'shock lung' state resulting from the embol that induces DIC) However, it can shorten clotting time and induce fibrinol,

There is little time for confirmatory tests The infusion is usually mass and over half the patients die at the time of the original incident Most of cases are confirmed at autopsy with findings of edema, alveolar ' and emboli (made up of fetal squames hair fat and mucin) plugging arterio of less than 1 mm in diameter A high index of suspicion is essential if patient who survives the initial embolic episode is to be saved Apparentl normal obstetric patients who suddenly go into profound shock and exhibi evidence of a coagulation defect, particularly immediately after delivery, be presumed to have had an amniotic fluid embolism

In survivors of the initial epsode, an electrocardiogram usually shows ev dence of right heart strain and a radiograph of the chest may show perih la infiltrates In 1947, Gross and Benz reported that aspiration of blood fror the right ventricle may confirm amniotic fluid embolization The blood ma separate into 3 layers, with amniotic debris floating over the buffy coat c white cells Most physicians are understandably reluctant to perform thi procedure in a severely ill patient but it may promptly confirm the diagnosi so that more vigorous treatment is undertaken In 1966 Altchek and Litwal suggested the use of lung scans using macroaggregates of ^{131}I albumin to demon strate perfusion defects and help establish the diagnosis in surviving patients However, it was not until 1973, when Gregory and Clayton again reported or the use of this technique, that the method became more widely accepted

Coagulation studies performed in these patients usually confirm the presence of DIC Coagulation factors are rapidly depleted, fibrinogen platelets, and factors V, VIII and XIII are particularly affected The activation of the fibrinolytic system is instantaneous and patients may continue to bleed after the intravascular coagulation has been controlled by heparin replacement with fresh blood, and fresh frozen plasma administration The delayed onset of embolism in some patients is probably due to the amniotic fluid being trapped in the uterine veins after delivery, and being gradually released into the circulation with the diminution of uterine tone

MANAGEMENT The treatment of patients with amniotic fluid embolism should have two objectives to support the cardiovascular system and to control the coagulopathy

Support the Cardiovascular System This subject is discussed elsewhere in this chapter Obviously, if the patient is bleeding profusely, administration of fresh whole blood in sufficient quantities to maintain an effective circulating blood

volume is essential If fresh whole blood is not available, then replacement with bank blood, platelet packs, and fresh frozen plasma should be carried out when there is evidence of severe coagulopathy and hypovolemia

Control the Coagulopathy Gregory and Clayton recommend the administration of heparin immediately upon clinical suspicion of amniotic fluid embolism They follow the initial recommendation of Reid and Weiner (1953), who advised administering 50–70 mg of heparin intravenously to help neutralize the effects on the clotting system of the amniotic fluid entering the circulation It seems unlikely that this dosage would produce any alterations in the thrombin component of the clotting mechanism The heparin should be given within 10 min after evidence of respiratory distress in any patient suspected of having had an amniotic fluid embolism If the blood fails to clot after heparinization, fresh blood, fresh frozen plasma, cryoprecipitate, or fibrinogen should be given The use of antifibrinolytic agents such as epsilon aminocaproic acid (EACA) should be reserved for patients who show laboratory evidence of fibrinolysis in spite of adequate control of DIC, and heparin should be given with them In pregnant patients primary fibrinolysis is rare, and secondary fibrinolysis is part of the normal defense mechanism, so it is rarely necessary to use EACA

SEPTIC SHOCK (ENDOTOXIC SHOCK)

Endotoxic shock is a syndrome resulting from sepsis caused by gram negative bacteria The noxious agent is a lipopolysaccharide protein complex liberated from the bacterial wall on the death of the organism The most common organisms involved are *Escherichia coli, Klebsiella-Enterobacter, Proteus mirabilis* or *vulgaris* and *Pseudomonas aeruginosa* The results of blood cultures obtained in a typical series of patients with endotoxic shock are presented in Table 3-3 Endotoxic shock in the young patient is relatively rare When it does occur, it is usually related to a pregnancy The most common cause is infected abortion, but it is also seen with chorioamnionitis and pyelonephritis

When shock results from infection, it is called septic or bacterial shock By usage, this term is now applied to the characteristic clinical picture produced by gram-negative infection and is frequently called gram negative shock or endotoxic shock.

TABLE 3-3 *Organisms Isolated from Blood Cultures Obtained from patients with Septic Shock*

Organisms Isolated	Patients (%)
E coli	35
Klebsiella Enterobacter	17
Proteus	15
Pseudomonas	19
Others	23

*Blood cultures were negative in 46% of patients with clinical septic shock.

The importance of septic shock is well recognized The mortality of septic shock in the United States was reported as 50% by Petersdorf in … and as 50–80% by Barnett and Sanford in 1976 Mortality in reported … ranges from 11 to 82% The variation in mortality is attributable to d… in patient type, in the underlying disease process, in the promptness of … and in the methods of management

The relatively young pregnant—or recently pregnant—patient with sep… shock would be expected to respond more readily to therapy than an olde… debilitated patient, and this has been the general experience However e… in these relatively healthy young people the mortality is considerable, and … covery is achieved only by prompt diagnosis and aggressive management

Pathophysiology

Wise and associates (1952) were among the first to report shock, withou… blood loss, in gram negative sepsis The correlation of vascular collapse … septic abortion with gram negative sepsis was made by Studdiford and Dougla… (1956) Their association of the two conditions was based on the work o… Good and Thomas (1952), who had produced shock in conjunction with bi… lateral renal cortical necrosis by repeated injection of sublethal doses of endo… toxin

For the past decade two principal mechanisms have been invoked to expla… the findings in endotoxic shock 1) selective vasospasm, and 2) disseminated intravascular coagulation (DIC) Selective vasospasm in small arteries and vein… was proposed by Lillehei and MacLean (1958) to explain the hemorrhagic necrosis observed in the intestines of dogs that died from the effects of endo… toxin Their observations led them to suggest that vasopressors should not be used in shock from endotoxin but that glucocorticoids should be beneficial because of their vasodilator effects Studies of peripheral resistance in patients with endotoxic shock however, have yielded conflicting data, with depression, elevation, or marked variation having been reported, perhaps depending upon the degree of shock when the studies were carried out

Disseminated intravascular coagulation was proposed by McKay *et al* (1959), because of the correlation between autopsy findings in patients who died of endotoxic shock and those seen in the generalized Shwartzman reac… tion in experimental animals, as reported by Good and Thomas (1952)

Since conclusive animal data that could be applied in the clinical situation are lacking, attempts have been made to clarify the mechanism of endotoxic shock by studies in primates The baboon is a very suitable animal for such studies inasmuch as its coagulation profile closely resembles that of the human and the animal is large enough that hemodynamic and biochemical changes can be measured with accuracy and relative ease In studies reported by Cavanagh *et al* in 1970, a single lethal intravenous dose of potent coliform endotoxin was used

From studies such as this it has become apparent that selective vaso… spasm, disseminated intravascular coagulation and reduced myocardial response to sympathetic stimuli all play an important part in the pathogenesis of endotoxic shock in the primate Other studies have revealed that the kidney is a primary target organ This helps to explain why acute tubular necrosis (ATN) is more commonly seen with this type of shock, rather than with shock from postpartum hemorrhage

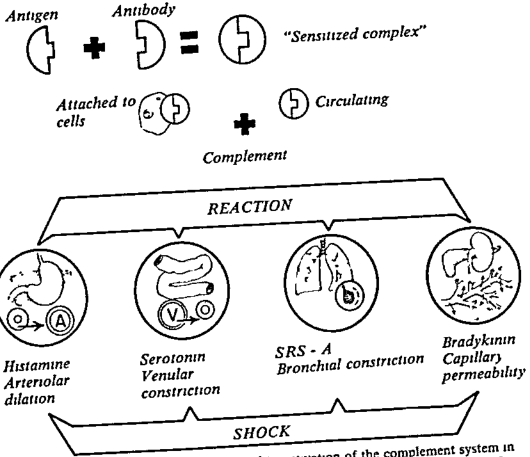


FIG 3-3 Some of the changes attributed to activation of the complement system in shock (Weil MH, Shubin H Diagnosis and Treatment of Shock Baltimore, William & Wilkins, 1967)

The lung deserves a great deal of attention in septic shock, because respiratory failure is the most common cause of death and may occur after the hemodynamic abnormalities have apparently been corrected The respiratory condition has been called "shock-lung syndrome," and is characterized by pulmonary congestion, hemorrhage, edema, capillary thrombi, and atelectasis Pulmonary surfactant decreases, and pulmonary compliance becomes progressively compromised

The effects of endotoxin on the individual cell are now receiving a great deal of attention At the cellular level, endotoxin causes damage by membrane effects and metabolic changes Experimental data suggest that ionic transport across membranes is affected early in the evolution of the shock state There is evidence that uncoupling of oxidative phosphorylation occurs somewhat later, with the inhibition of adenosine triphosphate (ATP) synthesis Metabolic processes such as lactate utilization that require ATP are inhibited, compounding the functional disarray of the cell

Many of these changes can be attributed to the activation of the complement system (Fig 3-3), but the exact mechanism remains in doubt It appears that both the classic and alternate (properdin) pathways are activated and

that complement activation is an important factor in cell injury Complement activation is known to result in the development of functional "holes in the cell membrane, and with the loss of control of permeability, cellular swelling and lysis occur Because most deaths occur from the "shock lung syndrome," it is also important to note that total complement depletion blocks the tendency to increased alveolar capillary permeability following endotoxin administration

Direct effects of endotoxin on the cell also play a role, as does lysosomal enzyme release The evolution of the process of cellular damage is further accelerated by the changes in ionic gradients that occur

In therapeutic studies, the value of administration of glucocorticoids and low-molecular weight dextran has been demonstrated Aspirin in clinical dosage also appears to protect against coliform endotoxin

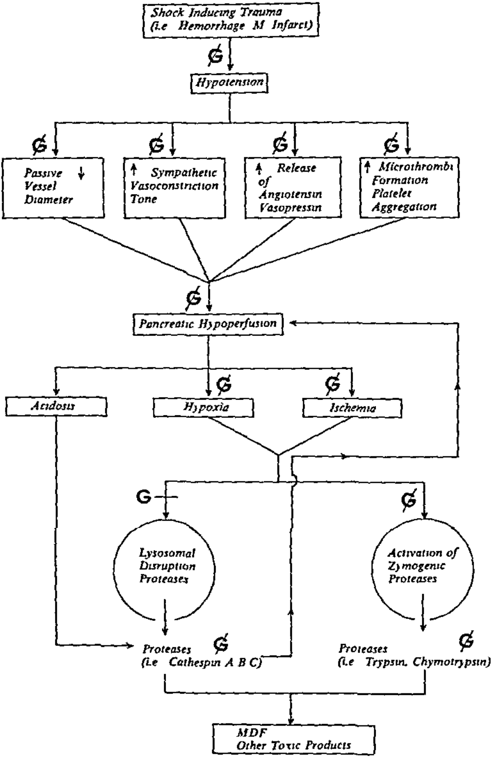
As a response to gram negative sepsis, cardiac output may temporarily increase The blood flow to the area of sepsis may be doubled or even tripled The rest of the increase in cardiac output is accommodated in the renal and splanchnic circulation It is generally accepted that there is a vasodilator substance released from the area of sepsis which decreases the resistance to flow through these areas Thus, when the patient goes into shock, the blood volume may be relatively normal, and the velocity of blood flow through certain areas may be increased

With endotoxemia, which may be a persistent state until the source of infection is removed or controlled, there is severe vasospasm of the postcapillary resistance elements This results in an excessive amount of blood being pooled in the capillary space as the precapillary resistance elements are in a state of vasodilation There is an increase in hydrostatic pressure, and capillary wall injury occurs secondary to it or as a direct consequence of the endotoxin itself Fluid may then escape from the intravascular to the extravascular compartment Nonnutritional channels of flow (shunt flow) are opened up secondary to the postcapillary venous obstruction Disseminated intravascular coagulation is initiated by the capillary wall injury, the general anoxemic state and/or by platelet injury secondary to the action of endotoxin Platelet aggregates and fibrin thrombi are deposited as the coagulopathy is established thus contributing to the obstruction to the flow of blood For a detailed discussion of DIC, see Chapter 2, Clotting Disorders in Pregnancy

The net result of all these factors is a reduction in the circulating plasma volume and in the venous return to the heart The patient may not have lost any blood, but a significant proportion of her blood volume is trapped in limited areas of her body, so that her circulating blood volume is reduced Nonnutritional shunt flows contribute to the overall state of underperfusion in certain organs According to Lefer and Glenn (1974), endotoxin may cause liberation of a myocardial depressant factor (MDF) from the pancreas that results in diminished contractility of heart muscle and a relative bradycardia This may be an important factor in the development of irreversible shock (Fig 3-4) In a circulation previously adjusted to the septic state, the fall in venous return plus the inability of the heart to compensate adequately will result in hypotension As the precapillary sphincters are in a state of vasodilation, the patient in early shock feels warm to touch and is said to be in the warm hypotensive phase of septic shock

With the development of hypotension the previously mentioned compensatory mechanisms of the body go into effect The 'sympathetic squeeze" is

FIG 3-4 Schematic representation of factors and conditions present in circulatory shock, which are involved in the formation of myocardial depressant factor (Lefer AM, Glenn TM Corticosteroids and the lysosomal protease myocardial depressant factor system in shock In Glenn TM (ed) Steroids and Shock Baltimore Univ, Park Press Copyright © 1974 p 233)



employed to shunt blood flow to the vital organs This sympathetic discharge has to be of great magnitude to override the effects of sepsis on the microcirculation, particularly if the endotoxemia is allowed to continue As peripheral resistance is increased by contraction of the arterioles and metarterioles the patient s skin becomes cold, clammy, and ashen gray This is the cold-hypotensive phase of septic shock

Much as in late hypovolemic, hemorrhagic shock, if therapeutic measures are not successful in controlling the endotoxemia the vascular elements become exhausted from the prolonged vasoconstriction and eventually perhaps when the pH has fallen to a critical level, they yield to the vasodilating effects of vasodilator substances released by injured tissue This results in secondary or irreversible shock with stagnant hypoxia vital organ damage, and death

The kidneys seem to be at particular risk in cases of septic shock Many of these patients exhibit oliguria and may develop acute tubular necrosis or, more rarely, bilateral renal cortical necrosis Thus, renal failure may be a late cause of death even if the patient survives the shock episode Initially renal blood flow is increased secondary to sepsis It may be this reactive hyperemia that predisposes the kidney to vascular injury from endotoxemia It has been demonstrated that the fall in renal blood flow occurs before any fall in aortic pressure and cardiac output, and that it is probably due to deposition of platelet aggregates and fibrin thrombi in the renal vascular afferent arterioles and glomeruli Irrespective of the reason it is a fact that the kidneys are in great danger in endotoxin shock, so the appearance of oliguria in a normovolemic patient is a bad prognostic sign

The nature of the pathophysiology of septic shock poses the following therapeutic implications 1) If the initial postcapillary resistance elements of constriction and capillary wall injury can be prevented then the shock state can be avoided or it may be mild Glucocorticoids produce both of these effects when given in pharmacologic doses 2) The use of vasodilators like phenoxybenzamine or isoproterenol may reduce the muscular venule vasoconstriction and the secondary sympathetic discharge 3) Blockade of the sympathetic system with drugs like reserpine or chlorpromazine may also be helpful 4) Platelet sparing drugs, such as steroids or low molecular weight dextran may reduce platelet aggregation 5) Heparinization may prevent further disseminated intravascular coagulation by its antithrombin action

The production of endotoxemia is an ongoing process and control of the infection is crucial for the patient The proper choice of antibiotics, used in large doses intravenously, is essential It is also important to bear in mind that the uterus is not essential for the survival of the patient If the patient with an infected abortion or severe postpartum endometritis is not improved by the use of antibiotics and removal of the septic focus by dilation and curettage then hysterectomy is the next logical step in controlling the infection

Clinical Aspects

PREVENTION Because mortality in endotoxic shock is high the occurrence of this condition should be prevented whenever possible

Patients with a diagnosis of infected abortion should be vigorously treated with appropriate antibiotics, and the septic focus should be removed early Large doses of penicillin (aqueous penicillin G, 30–60 million units per day in

intravenous fluids) or ampicillin (1 g intravenously every 4 hours) and gentamycin (1 mg/kg body weight intravenously every 8 hours) usually provide adequate therapy, if residual infected products of conception are present Oxytocin may also be used to aid in expelling products of conception, using 20 units in a liter of intravenous fluid If the infection follows elective infusion of saline into the amniotic sac, curettage is necessary after delivery of fetus and placenta An elevation of temperature over 102° F should prompt close observation for the development of endotoxic shock The potential for endotoxic shock in the patient with an infected abortion should always be kept in mind, although we have seen it in only 3 2% of such patients In other reports, the figure ranges from 2 5 to 6 4%

Chorioamnionitis This can frequently be prevented by oxytocin induction following spontaneous premature rupture of the membranes Induction should be performed, even in the afebrile patient, if there is no contraindication and the estimated fetal maturity is more than 36 weeks Induction may also be indicated when the estimated fetal maturity is 32–36 weeks, because the intrauterine hazard of infection may be greater than the extrauterine problem of prematurity A gram stained smear from an amniocentesis specimen may be useful in deciding whether intrauterine infection is present Individualization is necessary in these cases, however, and it should be remembered that the incidence of chorioamnionitis is higher among patients in the lower socioeconomic groups

Pyelonephritis In pregnancy pyelonephritis is mainly the result of urinary stasis due to mechanical pressure on the ureters by the uterus Renal intracalyceal pressure can be reduced, therefore, by having the patient lie on her side Although the disease is bilateral, the right side is usually more affected, and the patient is usually more comfortable lying on her left side Antibiotic therapy in adequate amounts is essential in this situation The choice of antibiotics is difficult during pregnancy since certain agents—*i e* tetracycline, sulfisoxazole, and the nitrofurantoins—have been reported to produce adverse effects on the fetus It is desirable to admit the patient to the hospital and give ampicillin, 1 g intravenously as a bolus every 4 hours, unless the urine culture reveals an ampicillin resistant organism

HISTORY Careful interrogation of the patient is essential Patients who have undergone illegal abortions, and even a few who have had legal abortions, will frequently give a clinical history that is vague or even contradictory Frequently, the menstrual history is entirely falsified Patients with chorioamnionitis should be asked about time of rupture of membranes and onset of symptoms In pyelonephritis, the time of the onset of flank pain, dysuria, and chills, as well as the estimated date of confinement, are important historic facts Because postpartum endometritis is often associated with manual exploration of the uterus, and even more commonly with intrapartum chorioamnionitis, a history of these should be sought

PHYSICAL EXAMINATION The patient is usually febrile, with a temperature in excess of 102° F Normal or subnormal temperature in association with shock is a grave prognostic sign warranting early aggressive management Local findings depend upon the site of infection In the patient with an infected abortion

there is suprapubic tenderness, and local or generalized rebound tenderness is frequently found. On speculum examination the cervix is bluish and soft, tenaculum marks may be visible. The external os may exude a foul smelling discharge, which should be cultured aerobically and anaerobically at once. Products of conception may be found in the cervical canal or vagina. Bleeding may be minimal if the products of conception have been expelled or removed. Bimanual examination reveals marked tenderness. The internal os may admit one finger. The uterus is enlarged and soft, and manipulation of the cervix or body of the uterus may elicit excruciating pain. Broad ligament tenderness is evidence of parametritis and pelvic cellulitis. Rarely, thrombosed veins are palpable.

In chorioamnionitis the findings are similar, with evidence of local or generalized peritonitis and a markedly tender uterus. Fetal heart tones are sometimes absent in this condition.

Postpartum endometritis is usually associated with a subinvoluted, tender uterus and signs of peritonitis.

In pyelonephritis, costovertebral angle tenderness will usually be elicited.

When first seen, the appearance of the patient is a useful guide to the type of treatment required. From a clinical viewpoint, septic shock may be classified as follows: 1) primary shock (reversible shock) of either the early (warm hypotensive) phase, or the late (cold hypotensive) phase, 2) secondary shock (irreversible shock) (Table 3-1).

Primary Shock—Early (Warm Hypotensive) Phase. In this phase the patient is hypotensive, alert, and apprehensive. Her face is flushed, and her skin is warm. Temperature is usually in the range of 101°–105° F (38.4°–40.6° C). Profuse sweating is not uncommon. A shaking chill coinciding with the temperature peak may be seen. There is usually a moderate tachycardia, 100–110 beats/min, but about 20% of patients have a pulse rate under 72 beats/min. The pulse pressure remains satisfactory, and the urinary output is good at this stage.

Primary Shock—Late (Cold Hypotensive) Phase. In this phase of primary shock, the patient is hypotensive, pale, and clammy. The temperature itself is often subnormal. She gradually becomes less alert and less apprehensive. As the blood pressure drops, oliguria may supervene. The triad of hypotension, tachycardia, and oliguria is typically present in this phase.

Secondary Shock (Irreversible Shock). In this phase the patient is cold and clammy with an ashen cyanotic appearance. Anuria, cardiac or respiratory distress, and coma are grave prognostic signs. Most patients die because of the development of severe shock-lung syndrome.

MONITORING THE PATIENT. Careful monitoring of the patient in endotoxic shock is essential; the clinical measurements most useful in our experience are presented in Table 3-4.

The pulmonary artery wedge pressure, which reflects left ventricular end diastolic pressure, is a more useful guide for volume replacement than the central venous pressure (CVP), but Swan Ganz catheters are not universally available.

TABLE 3-4 Clinical Measurement for Monitoring Endotoxic Shock

Clinical measurement	Frequency of intervals (min)
Pulse rate	15
Blood pressure and pulse pressure	15
Central venous pressure (or pulmonary artery wedge pressure)	30-60
Urinary output	60

No single parameter is adequate alone Moreover, though urinary output is a good guide for the immediate care of a patient in shock, the finding of a urine/serum osmolality ratio over 1 5 is a more reliable indication that hypovolemia is the problem rather than acute tubular necrosis This is useful as a monitoring measure in the further care of a patient with acute renal failure (See Chapter 5 Acute Renal Failure) as a complication of pregnancy

Ancillary studies should include the following

Complete blood count
From endocervical or intrauterine swab
1. Gram-stained smear
2. Culture and sensitivity tests
From indwelling Foley catheter
1. Urinalysis
2. Gram stained smear
3. Culture and sensitivity tests
Blood cultures at time of temperature peaks
Serum electrolytes, blood urea nitrogen, and uric acid
Electrocardiogram
Chest x-ray for pneumonitis or infarction
Lung scan if infarction suspected
Abdominal x ray for foreign bodies, gas pattern, or free air
Blood coagulation profile for evidence of DIC
Arterial blood lactate
Arterial pH, Pa_{O_2} and Pa_{CO_2}
Blood volume (early shock)

These studies should be repeated as necessary

Blood volume measurements are unreliable in the later stages of endotoxic shock, because of circulatory stagnation and sequestration of fluid

MEDICAL MANAGEMENT The success of treatment will depend largely upon the promptness of diagnosis Every infected patient must be observed closely for the early signs of shock Treatment must be tailored to the needs of the individual patient When a removable septic focus is present, surgery is the keystone of treatment When the septic nidus cannot be removed, as with pyelonephritis, for example, therapy can only be medical

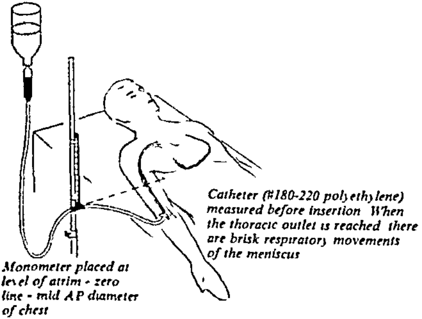
The essential steps in management are as follows

1. Ensure that the patient has an adequate airway If necessary, an endotracheal tube should be passed or a tracheostomy performed
2. Adequate fluid and blood replacement should be carried out, using as guides the CVP (Fig 3-5), or better still, pulmonary wedge pressure using a Swan-Ganz catheter (Fig 3-6), the urinary output and the blood volume estimation Blood, plasma, 5% serum albumin, or 5% dextrose in saline should be given as indicated Low molecular weight dextran is useful because it gives volume replacement and reduces sludging in the microcirculation, but its value in these respects must be weighed against the tendency to cause a coagulation defect

   Cardiac competence can be tested in a patient with a borderline CVP (12–16 cm H_2O) by infusing 500 ml 5% dextrose in water or 5% serum albumin at 20 ml/min If at the end of the infusion the CVP has not increased more than 5 cm H_2O, and it falls to within 2 cm of the preinfusion level, then cardiac competence can be assumed, at least for the present time

   Metabolic acidosis is common and becomes progressive if not corrected, in this case, 0 45% saline, with one (44 6mEq) or two ampules of sodium bicarbonate added, is useful Sodium bicarbonate acts rapidly and provides

FIG 3 5 A basilic or brachial vein cutdown at the elbow is performed and a large polyethylene catheter is passed as far as the superior vena cava or until wide excursions in central venous pressure occur with each breath. Zero reference point for measurements is the middle of the anteroposterior diameter of the chest Central venous pressure can also be measured by direct puncture into the subclavian vein under and parallel to the first portion of the clavicle (From Thal AP, Wilson RF In Current Problems in Surgery Chicago Year Book Medical Publishers, Copyright © 1965 Used with permission)



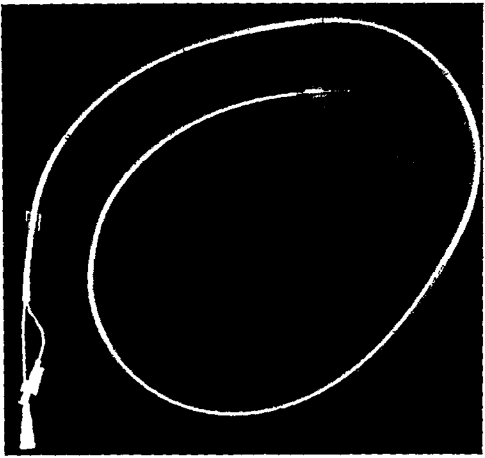


FIG 3-6 Balloon flotation catheter (Swan Ganz) Catheter is inserted into external jugular or subclavian vein The balloon is inflated with 0 8 ml of air It is carried in the bloodstream through right atrium and right ventricle, eventually wedging in one of the smaller branches of the pulmonary artery Pressures can be measured which accurately reflect left ventricular end-diastolic pressure

good buffering action Lactate should not be used for correction of acidosis in these patients, because conversion to bicarbonate requires aerobic metabolism

3. *Antibiotics should be selected according to the organisms found on the* Gram-stained smear, and on the basis of the previously known antibiotic sensitivities from similar organisms in the hospital in which the patient is being treated The intravenous route and massive dosage must be employed Penicillin (or ampicillin) and chloramphenicol are the most generally useful drugs, but gentamycin is required to combat *Pseudomonas* infection Penicillin should be given as crystalline penicillin G in a dosage of 10 million units every 4 hours in intravenous fluids As an alternative to penicillin, ampicillin 1–2 g every 4 hours intravenously may be used Chloramphenicol is given as an intravenous bolus, 1 g in 100 ml saline every 6 hours intravenously may be used In addition gentamycin 1 3 mg/kg body weight should be given

intravenously every 8 hours Nephrotoxic drugs should be avoided in the presence of oliguria

Most crystalline penicillin is supplied in the form of the potassium salt If this is being used, 60 million units of potassium penicillin G contains 90 mEq of potassium, which may become a hazard in the presence of renal failure Because ampicillin is in the form of the sodium salt, it should be used in preference to penicillin under these circumstances Also, in the presence of renal failure gentamycin should be given on the basis of the following formula

Serum creatinine (mg/100 ml) × 8 = frequency of administration (in hours)

It has been suggested that bactericidal drugs should not be employed in septic shock because massive destruction of organisms could lead to massive liberation of endotoxin This idea, however, is not borne out by experience in clinical practice

4 Glucocorticoids should be given in pharmacologic doses and appear to be useful (Table 3-5) Schumer (1976) has recently reported on their efficacy in a controlled study Methylprednisolone sodium succinate (Solu Medrol), 30 mg/kg body weight per day, or dexamethasone (Decadron), 6 mg/kg of body weight per day, should be given by continuous intravenous infusion after a loading dose of methylprednisolone sodium succinate (125 mg) or dexa methasone (20 mg) According to Sladen (1976), four times this dosage may be required for the shock lung syndrome These agents probably exert a beneficial effect in at least four ways by stabilizing lysosomes at the cellular level, by their inotropic action on the heart, by improving renal perfusion and by maintaining the blood volume Abrupt discontinuance after up to 72 hours apparently produces no ill effects, so there is no need to follow a regimen of gradual withdrawal

5. Vasomotor drugs should be given as indicated by the clinical state of the patient In septic shock, the primary aim of therapy is improvement of tissue perfusion rather than the restoration of normal blood pressure It may seem more knowledgeable to talk of alpha mimetic agents, beta-mimetic agents, alpha blockers and beta blockers but the fact is that all these agents have been used successfully in the management of experimental endotoxin shock All these agents have an action on the myocardium and on the

TABLE 3-5 Mortality in Steroid Untreated Versus Steroid-Treated Patients with Septic Shock Over the Period July 1 1959 to June 20, 1976

	No of deaths per total No of patients		
Group	No. steroids	Steroids	Total
A (1959-65)	3/6	0/12	3/18
B (1966-76)	0/0	4 43	4/43
Total	3/6	4/55	7/61

peripheral vascular bed Since the choice of drug will depend largely on the patient's appearance, it is more clinically germane to talk of vasopressor and vasodilator drugs In the warm-hypotensive phase, a vasopressor rather than a vasodilator drug is indicated We have found metaraminol (Aramine) to be the most useful of the vasopressor drugs in the management of this condition It is given only in sufficient quantities to maintain systolic blood pressure at the lower limit that ensures an adequate urinary output

When a patient is seen in the cold-hypotensive phase, however, generalized vasoconstriction is present, and there is usually also an element of hypovolemia Volume replacement, and vasodilator drugs in small doses, is the treatment of choice at this stage Chlorpromazine (Thorazine), 5–10 mg intravenously every half hour, as needed, is the safest vasodilator When the central venous pressure is elevated, and the pulse rate is in the normal range, isoproterenol (Isuprel) may be used for its inotropic and vasodilator (beta-receptor) effects Because of its tendency to produce cardiac arrhythmia, it should not be used in the presence of a pulse rate in excess of 120 per minute

i. Digitalization should be carried out when the central venous pressure uses above 16 cm H_2O and tachycardia is present If the serum potassium level is normal, lanatoside C (Cedilanid) 0 8 mg as an initial dose should be followed by 0 4 mg every 6 hours twice A maintenance dose of digoxin, 0 25 mg daily, is then given and the patient is monitored by ECG and blood digoxin determinations if available

Other measures are as follows Heparin administration should be considered f clotting studies indicate the presence of a consumption coagulopathy—viz, hrombocytopenia, hypofibrinogenemia, a fall in fibrinogen factors V, VIII, and XIII, and the presence of fibrin split products If a full coagulation profile is not available and the patient is not responding to the standard treatment cited in the six steps listed, then heparin should be tried Heparin should not be used simply as a routine There is no evidence that hypothermia or hyperbaric oxygen are of value in endotoxic shock More recently l-dopamine, 200 mg in 500 ml 5% dextrose in water, given as an infusion, and titrated against the blood pressure, has been advised This is a very useful drug but must be used with care

SURGICAL TREATMENT When septic shock is associated with a surgically removable nidus of infection, the focus must be removed In the woman with a septic abortion the nidus should be removed within 6 hours of diagnosis, provided adequate supportive measures are underway Usually removal can be accomplished by dilation of the cervix and evacuation of the uterus with ring forceps, followed by suction and sharp curettage However, when the uterus is over 14 weeks in size, complete evacuation by curettage is difficult and infected tissue may be left behind (Fig 3-7) Also, when the disease has advanced to the stage of microabscess formation in the myometrium, hysterectomy is the only logical surgical treatment (Fig 3-8)

Hysterectomy should be considered if 1) the patient continues in shock following curettage and adequate supportive measures, 2) the uterus is more than 14 weeks' in size, 3) the uterus is perforated, 4) the patient is oliguric, 5) intrauterine *Clostridium welchii* infection is diagnosed, or 6) a corrosive or toxic douche has been used The technique of hysterectomy should be tailored to fit the particular problem For example, in the presence of clostridial infec-

FIG 3-7 Hysterectomy specimen demonstrating presence of unsuspected infected retained products

tion, pedicle size should be kept small, so that a minimum of devitalized tissue is left behind

Septic pelvic thrombophlebitis with involvement of both ovarian and hypogastric vessels is not uncommon in patients with long-standing infected abortions, postpartum endometritis, or chorioamnionitis Septic pulmonary embolization may occur, and coalescing lung abscesses may be the ultimate cause of death

Inasmuch as the underlying problem is septic rather than thrombotic, anticoagulation is contraindicated unless a special problem exists Ligation of the inferior vena cava and ovarian veins is the treatment of choice, although heparin may be tried when aseptic thrombosis is suspected The transperitoneal route should be used to gain access to the ovarian veins Collins (1970) has

FIG. 3-8. Microabscess formation in the myometrium following septic abortion

reviewed the use of this operation in his extensive report on suppurative pelvic thrombophlebitis. Some have suggested that septic thrombosis can be treated *successfully with heparin, but as pointed out by Gibbs (1976), in the patients* treated "successfully," the diagnosis has been a presumptive one.

If vaginal delivery is not accomplished within 12 hours of the onset of chorioamnionitis, abdominal delivery should be seriously considered. It is best *accomplished by low transverse cesarean section. If severe infection or en-*dotoxic shock is present, cesarean hysterectomy should be performed. Ligation of the inferior vena cava and ovarian veins should be carried out if there are palpable clots in the hypogastric or ovarian veins or if the patient has had a pulmonary infarction In an occasional patient with persistent anuria, dialysis may be required. In this case, consultation with a nephrologist is indicated (see Ch. 5, Acute Renal Failure as a Complication of Pregnancy).

CARDIOGENIC SHOCK

CARDIAC ARREST

Sudden failure of the pumping action of the heart will lead to an immediate reduction in available blood volume. This can be caused by such conditions as myocardial infarction, cardiac tamponade, or massive pulmonary embolism,

but when it occurs in an otherwise healthy woman, it is usually referred to as "cardiac arrest" A common cause of the latter is anesthesia Cardiac arrest now occurs about once in every 5000 instances of anesthesia administration (see Ch 13, Anesthetic Emergencies)

MYOCARDIAL INFARCTION

The diagnosis is established from the patient's history and electrocardiographic evidence of myocardial ischemia An x ray film of the chest may show evidence of cardiac enlargement with pulmonary edema Elevated levels of transaminase, in the absence of liver, pulmonary, or muscular damage, are also helpful in establishing the diagnosis The patient is usually cold and clammy with a low blood pressure If there is concomitant right heart failure, the central venous pressure remains high

Discussion of detailed management of a patient with myocardial infarction can be found in many medical textbooks and will not be dealt with here

CARDIAC TAMPONADE

This type of cardiogenic shock is the result of an effusion into the pericardial sac, which interferes with diastolic filling of the heart and may result in a critical reduction in cardiac output, a fall in arterial blood pressure, and a reduction in tissue perfusion Pericardiocentesis is the definitive treatment and supportive therapy is also required For details of management the reader is referred to the many medical textbooks on the subject

PULMONARY THROMBOEMBOLISM

Pulmonary infarction secondary to thromboembolism is one of the most common causes of maternal death In 50% of cases the diagnosis is made only postmortem

Pulmonary thromboembolism can occur without any obvious evidence of venous thrombosis in the veins of the pelvis or the lower extremity If the thrombosis results in partial vein occlusion, few local symptoms may occur Current studies using ^{125}I labeled fibrinogen scanning in patients after operation have identified silent venous thrombosis of the lower extremities in approximately 35% of them This is understandably frustrating because thromboembolism can be a preventable disease if the susceptible patient and the onset of the initial venous thrombosis can be identified clearly Although pulmonary embolism may occur at any time during pregnancy, labor or the postpartum period it most commonly occurs during the first ten days of the puerperium Thromboembolism is more common following cesarean section and in patients given estrogens for the suppression of lactation

Pathogenesis

In 1854, Virchow pointed to a triad of factors contributing to the formation of venous thrombosis These are still the important ones today and are 1) alteration in the coagulation factors of the blood, 2) trauma to the vessel wall, and 3) venous stasis

Changes in blood coagulation in the postoperative or postpartum state include an increase in some of the intrinsic coagulation factors, especially factors VIII, IX, and X There is also an increase in fibrinogen, circulating fibrinolysin inhibitors, and platelet adhesiveness and aggregation in the 72 to 96 hours following surgery, and in the ten days after delivery Tissue necrosis from the surgical procedure may result in the release of thromoplastin-like substances During the normal puerperium the platelet count peaks around the tenth day

There are extensive venous collaterals in the female reproductive organs Postpartum or postabortal pelvic infection contributes to setting up the nidus for venous thrombosis It is obvious that the enlarging uterine mass in pregnancy results in diminished venous return from the lower extremities and the pelvic veins Venous stasis is extremely important in the production of venous thrombosis It results in platelet aggregation with adhesion to the vein wall, and the release of procoagulant substances that form an initial platelet, fibrin, and erythrocyte network This process eventually leads to thrombus formation The degree of postpartum immobilization is directly related to the incidence of thromboembolism, so early ambulation is important in the prevention of pulmonary embolization

Once venous thrombosis is established, the size of the clot increases by aggregation, and a long friable clot may result Pulmonary embolization occurs by fragmentation of this clot, or by detachment of the entire clot from the vessel wall If the embolus is small, the patient may suffer few symptoms and survive If a large portion of the clot becomes detached, a major branch of the pulmonary artery may be occluded This will cause reflex spasm of other branches of the pulmonary artery, and death frequently results, usually by cardiac arrest

Diagnosis

Pulmonary infarction often presents a problem in diagnosis This is unfortunate, for many patients die from a repeat episode while the physician debates the advisability of heparinization

A very high index of suspicion is necessary, and should apply especially to the type of patient who is at high risk for thromboembolic disease The following factors increase the incidence of pulmonary embolization in the pregnant woman age over 35 years, obesity, immobility or paralysis, pulmonary disease, cardiac disease, diabetes mellitus, malignancy, ascites, polycythemia, varicose veins, a previous history of thrombophlebitis, prolonged airline travel, and the use of hormones for the suppression of lactation If these high-risk factors are kept in mind, then the diagnosis of pulmonary embolism is more likely to be made in the patient who suddenly experiences chest pain or dyspnea Moreover, it is obvious that prophylactic measures should be taken in these high-risk patients to prevent the initial peripheral thrombosis

Clinically, small emboli may be asymptomatic When symptoms do occur, tachypnea or dyspnea are the earliest and most common ones If the embolus is large, there may be an increase in pulmonary artery pressure, which eventually will cause right ventricular strain or failure Return of blood to the left ventricle is impaired, and left ventricular output will fall The patient then exhibits the usual signs and symptoms of shock tachycardia, hypotension, coldness of the skin, and diaphoresis Cyanosis frequently accompanies pulmonary thrombo-

embolism Impairment of coronary blood flow may result in angina Diminished cerebral circulation produces confusion or syncope There is frequently a feeling of impending doom Hemoptysis and pleuritic chest pain occur later, after the segment supplied by the blocked artery is infarcted

On examination, the chest may appear normal or there may be a faint friction rub with rales In time, tenderness of the intercostal muscles overlying the area of pleural reaction may develop Neck veins are frequently distended, and the central venous pressure may be elevated as a result of right ventricular failure Pleural effusions are uncommon at an early stage but eventually may become quite massive Abdominal examination may reveal marked muscle guarding, that simulates the rigidity seen in acute abdominal conditions If iliofemoral thrombosis is present, the entire leg may be swollen and tender If popliteal or posterior tibial venous thrombosis is present, examination may reveal edema of the foot or calf, tenderness along the femoral vein, or calf pain on dorsiflexion of the foot (Homans' sign) Pain in the calf may be elicited in venous thrombosis by placing a sphygmomanometer around the thigh and pumping it to 160 mm Hg of pressure (Lowenberg's sign) Excessive pressure on the calf and repeated attempts to elicit Homans' sign may result in embolization and should therefore be avoided When pelvic examination may reveal the presence of uterine and parametrial tenderness consistent with pelvic infection, the possibility of septic pelvic thrombophlebitis must be considered

EMERGENCY DIAGNOSTIC TESTS FOR PULMONARY EMBOLISM A battery of tests suggested by Dolen and Dexter (1976) provides both a method of diagnosis and a guide to management (Table 3–6)

ROENTGENOGRAPHIC EXAMINATION X-ray films of the chest may be useful in confirming the suspicion of pulmonary infarction However, emboli frequently produce no shadows for days On the other hand, frank consolidation, pleural effusion, and elevation of the diaphragm may be seen Serial chest x rays are required for demonstration of progressive pulmonary parenchymal changes, and of development of dilatation of the main pulmonary artery and right ventricle All these changes, however, are nonspecific and may be seen in congestive failure, atelectasis, and pneumonitis

ELECTROCARDIOGRAM An ECG may be of help in the differential diagnosis of pulmonary and cardiac infarction In pulmonary embolism the ECG may be normal or merely show a sinus tachycardia In massive pulmonary embolism, it may show right axis deviation and right axis strain with peaked P waves and occasionally S–T segment changes indicative of right ventricular strain or ischemia Acute cor pulmonale is almost always due to pulmonary embolization, provided a recent previous ECG tracing is known to be normal

BLOOD CHEMISTRY STUDIES Both lactic dehydrogenase (LDH) and the serum bilirubin may be elevated, while the serum glutamic oxaloacetic transaminase (SGOT) level is usually normal A normal SGOT value 24–48 hours after the onset of chest pain almost always excludes myocardial infarction Szucs (1971) has shown that this trio of laboratory tests is diagnostic in approximately only 12% of known cases of pulmonary embolization Blood gas determinations

TABLE 3-6. Emergency Diagnostic Tests for Pulmonary Embolism

Test	Findings suggestive of pulmonary embolism	Aids to differential diagnosis	Therapeutic implication if pulmonary embolism is present
ECG	Right axis shift (S1Q3T3), right ventricular strain, and new incomplete right bundle branch block	To rule out acute myocardial infarction	Detection and treatment of arrhythmias
Chest roentgenogram	Enlargement of main pulmonary artery and right ventricle, infiltrate, pleural effusion, elevated diaphragm, or asymmetry of vasculature	To rule out pneumonia and congestive heart failure	Presence of acute right ventricular enlargement indicates life-threatening embolism
Arterial blood gases	Low PO_2 and PCO_2 are nearly constant findings in acute embolism	Normal PO_2 nearly excludes acute pulmonary embolism	Guide to oxygen therapy and guide to prognosis
Central venous pressure (CVP)	Elevated (if right ventricular failure is present)	If hypotension is present, low CVP, normal PA pressures nearly exclude pulmonary embolism as cause of hypotension	Central venous and PA catheters provide route for administration of drugs or fluids and ready access to blood samples
Pulmonary artery catheter	Increased PA systolic and diastolic pressures normal wedge pressure		
Lung scan	Segmental perfusion defects that ventilate normally	Normal scan excludes pulmonary embolism Scan may be equivocal	Extent of avascular areas serves as a guide to severity of pulmonary embolism
Pulmonary angiography	Finding defects due to presence of emboli cutoffs of pulmonary arteries	Normal angiogram excludes significant pulmonary embolism	Most accurate guide to extent of embolism

(Dalen JE, Dexter L. Pulmonary embolism In Weil MH, Shubin H (eds) Critical Care Medicine Hagerstown, MD, Harper & Row, 1976)

will reveal low P_{O_2} and P_{CO_2} in nearly all patients with acute pulmonary infarction, so a normal P_{O_2} almost completely excludes this diagnosis. Lung scanning with ^{131}I or technetium-labeled macroaggregates of albumin has increased the diagnostic accuracy in recent years. The method demonstrates abnormalities in the distribution of blood flow in the lung and areas of pulmonary artery obstruction. The evidence is only presumptive, as the procedure cannot differentiate between pulmonary embolization and other disease processes producing segmental decreased arterial flow, *i.e.*, pneumonia, atelectasis, pneumo-

thorax, carcinoma, or granulomatous disease However, it is more dependable than the chest x-ray and is a better screen test for pulmonary abnormalities if the patient's condition permits its use.

PULMONARY ARTERIOGRAPHY Recently, arteriography has been used to provide positive angiographic evidence of the extent and anatomic location of the pulmonary embolus 1) in patients who have equivocal findings suggestive of pulmonary embolization or 2) in patients with massive pulmonary embolism who may require immediate embolectomy Most authorities agree that pulmonary arteriography is the most definitive diagnostic study, and it should be employed whenever there is a serious question as to diagnosis The pulmonary artery pressure can also be measured by this technique As Bonnar has pointed out, the noninvasive techniques of ultrasonography, plethysmography, and thermography have not yet been adequately evaluated in pregnancy or the puerperium Laboratory methods such as estimation of serum fibrinogen or fibrin degradation products and plasma levels of beta-thromboglobulin are being investigated, but the nonspecific nature of these assays, especially in pregnancy, presents a difficult problem

Management

PREVENTION This consists of the identification of the patient who is at high risk for thrombophlebitis Mattingly and Wilkinson (1973) suggest that if prophylaxis against thromboembolism is to be achieved once the high risk patient is identified, prophylactic measures must be directed toward prevention of venous stasis in the lower extremities

In surgery, the most practical method of improving venous return from the legs is to elevate the latter 15° above the horizontal Thus when the patient is placed in the Trendelenburg position the legs should be kept straight rather than bent at the knee Cushioning the heel with a pillow on the operating table will prevent compression of the deep leg veins in the calf Several techniques, such as galvanic calf stimulation, encasing the legs in a plastic envelope with rhythmic alterations of pressure, and passive flexion and extension of the foot, have been shown to improve circulation in the legs during surgery

Various drugs such as heparin, sodium warfarin, dextran, and salicylates have been utilized in an effort to prevent surgical thrombophlebitis in the high risk patient Administration of low dosage heparin (5000 USP units 2 hours before surgery, and every 8 hours thereafter for 5 postoperative days) is an effective method of preventing postoperative venous thrombophlebitis in the patient who has undergone cesarean section or cesarean hysterectomy Gordon-Smith has reported that the incidence of venous thrombosis in the patient undergoing operation can be reduced to one-sixth the expected rate by preoperative heparinization Bleeding is not a problem because this dosage has a negligible effect on the clotting time of the patient The high-risk patient should be similarly treated even with a normal vaginal delivery

Once a patient develops a deep venous thrombosis, she should be given anticoagulants if they are not contraindicated The contraindications to anticoagulant therapy are as follows 1) blood dyscrasias, 2) ulcerative lesions of the gastrointestinal tract, 3) subacute bacterial endocarditis, 4) severe hyper-

tension with a history of encephalopathy, and 5) severe hepatic or renal disease

Our method of anticoagulation of the patient with deep vein thrombosis is to give 5000–10,000 units of heparin intravenously every 4–6 hours The dose is adjusted according to the clotting time or partial thromboplastin time (PTT), which is determined 30 min prior to administration of the heparin The goal is to maintain a clotting time two or three times normal A range of 40–60 sec in PTT usually indicates that therapy is satisfactory Around the fifth day the patient is started on an oral anticoagulant, usually sodium warfarin, and combined therapy is maintained for about 5 days At this time, the heparin is gradu ally discontinued and the patient is maintained on the oral anticoagulant for 6 weeks after symptoms of thrombophlebitis have subsided During this period the prothrombin time (PT) is checked twice a week and the dose is adjusted to keep the PT at two to two and one half times normal The patient is also kept at bed rest with the leg elevated until all symptoms have subsided

In cases of iliofemoral thrombosis immediate surgical therapy may be necessary to prevent propagation of the clot into the vena cava with consequent pulmonary embolization However, anticoagulant therapy should be tried first Even after the patient is anticoagulated, thrombectomy to remove the obstructing thrombus may still be required, and anticoagulation reduces the incidence of postembolectomy embolism It also reduces the incidence of the disabling postphlebitic syndrome

TREATMENT If an acute pulmonary embolism does occur, the initial and immediate treatment must be with intravenous heparin in a dose of 10,000–15,000 units (USP) This may help achieve immediate improvement of lung perfusion while the alveolar oxygen concentration is enhanced by the use of nasal oxygen or a respirator Arterial Po should be measured at once An increase in the arterial Po, within 30 min and a decrease in the respiratory rate indicate improvement and a more favorable prognosis If there is no improvement and the patient continues to go downhill, pulmonary embolectomy may be required as an immediate life saving procedure Other therapeutic adjuvants are as follows Morphine sulfate (10 mg) is usually given and repeated in 1 hour, if necessary, to allay pain and anxiety Rapid intravenous digitalization should be carried out with lanatoside C (Cedilanid) an initial dose of 0 8 mg intravenously followed by 0 4 mg every 4 hours to a total of 1 6 mg If the patient survives, she is started the next day on digoxin, 0 25 mg daily For hypotension metaraminol (Aramine), 100 mg in 5% glucose, is useful for its known inotropic effect, which improves coronary arterial perfusion and myocardial function Aminophylline, 250–500 mg intravenously, can be used to alleviate dyspnea

If the patient survives, anticoagulation with heparin is best continued as an intravenous infusion of 600 units per kilogram of body weight per 24 hours, altered in accord with clotting time or PTT An oral anticoagulant such as warfarin (Coumadin) is started after 1 week of heparin therapy The heparin is then tapered off over the next 72 hours Thereafter, the patient is maintained on sodium warfarin for a period of 6 months to 1 year at a dose level which decreases the prothrombin time to approximately 25% of the normal value When the embolus is thought to have originated in the legs, the patient is con fined to bed with the legs elevated to about 15° for about 5 days

NEUROGENIC SHOCK

ASPIRATION OF VOMITUS

This is the most common cause of maternal death from general anesthesia It can also occur in the course of high spinal anesthesia Aspiration of particulate matter causes atelectasis and perhaps ultimately lung abscess formation Aspiration of gastric secretions causes a severe chemical pneumonitis (Mendelson s syndrome) (see Ch 13 Anesthetic Emergencies for details)

SPINAL HYPOTENSION

High spinal anesthesia that causes respiratory paralysis and cardiogenic shock is the second most common cause of maternal anesthetic death This can occur with improperly given caudal or epidural anesthesia The basic problem is that the capacity of the vascular bed is suddenly increased with the production of relative hypovolemia. For discussion of the prevention and management of spinal hypotension see Chapter 13 Anesthetic Emergencies

INVERSION OF THE UTERUS

This condition is probably more common than is reported because most cases are the result of mismanagement of the third stage of labor The rate of occurrence is probably about 1 in 5000 vaginal deliveries

Predisposing Factors

1 Attempts to hasten the third stage of labor by combined traction on the cord and fundal pressure on a relaxed uterus
2 A thin walled hypotonic uterus with a patulous cervical canal
3 A fundal myoma
4 A previous history of inversion

Management

1 Shock must be combated with adequate blood transfusion oxygen and elevation of the legs as described earlier With inversion of the uterus shock is out of proportion to blood loss
2 The patient is deeply anesthetized
3 If the placenta is still attached to the uterine wall it should be removed and the uterus replaced in the normal position If the condition is recognized at an early stage this can usually be achieved by upward digital pressure on the inverted fundus while counter traction is applied using ring forceps on the anterior and posterior lips of the cervix.

   It is advisable to keep the hand within the cavity until the uterus is felt to contract under the influence of intravenous oxytocin
4 The uterus should be packed with gauze after replacement to help retain it in position

   When inversion is not recognized for several hours repositioning via the vagina may become impossible because of edema In this case Huntington s

operation is a simple abdominal curative procedure in which the inverted fundus is pulled up from the depth of the inversion by progressive traction with tenaculum forceps until the fundus is in the normal position Operative treatment should not be attempted until the patient is out of shock, unless hemorrhage is severe The serious nature of inversion of the uterus is appreciated by recalling that even among recognized and treated cases, the mortality is about 10%

ELECTROLYTE IMBALANCE

Severe loss of fluid or electrolytes may result in shock This may be seen in the diuretic phase of acute tubular necrosis, when inadequate attention has been paid to fluid and electrolyte replacement It is also common in traumatic shock because fluid is sequestered in the traumatized organs Often fluids must be given until the patient has gained 3 kg in weight in order to maintain an adequate central venous pressure In these patients, when homeostasis is established, excess fluid is lost by diuresis In the patient with shock, but without heart disease, fluids containing sodium are of paramount importance in expanding the intracellular fluid volume Sodium bicarbonate should be used in preference to Ringer's lactate, since the ability of the liver to convert lactate to bicarbonate is reduced in severe shock When a patient is in the late phase of primary shock with metabolic acidosis present, 5% dextrose in water with the addition of sodium bicarbonate is preferable to Ringer's lactate Isotonic sodium chloride may also be used for the expansion of the fluid in the extracellular space, but if a large volume is given without buffer it may potentiate the acidosis

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Life-Threatening Infections

Denis Cavanagh, Ralph E. Woods

Chapter 4

When I look back upon the past I can only dispel the sadness which falls upon me by gazing into that happy future when the infection will be banished

Ignatz Philip Semmelweis (1818–1865)

The dream of Semmelweis has not yet been fulfilled, and even today infection is one of the three most common causes of maternal death in the United States In addition, maternal infections and their treatment result in an increase in the stillbirth rate and in infant morbidity and mortality Two of the reasons for the persistence of infection as an important cause of maternal death have been the overdependence on antibiotics and the neglect of good aseptic technique Also to be considered is that many antibiotics used to combat life-threatening infections during pregnancy have not been approved for use in pregnancy by the Food and Drug Administration (USA), and so the benefit to the mother must be balanced against possible damage to the fetus

Although almost any infection can become serious in pregnancy, this chapter will concentrate on those infections that are most commonly lethal to the mother or her baby

Most of the life-threatening infections are now hospital-acquired Aerobes, such as *E coli*, enterococci and beta-streptococci, and anaerobes, such as *Bacteroides fragilis*, are the bacteria most commonly responsible Most bacterial infections are mixed, so two or three antibiotics are usually required for adequate control Although an infection may be fatal because of the virulence of the organism, as with *Clostridium perfringens*, mortality may result from failure to drain an abscess cavity or to remove a septic focus harboring more mundane organisms Viral infections such as influenza may be serious for the mother, and even a mild maternal herpes simplex infection may be fatal if transmitted to the baby

ANTEPARTUM INFECTIONS

The pregnant woman often develops complaints that may be explained by the altered physiology of normal pregnancy Time allotted to prenatal visits in a clinic or in a busy practice is usually short, and the examination may be performed totally or in part by an obstetric nurse Also, if the signs of impending infection are minimal, the problem may not be recognized until overwhelming sepsis occurs Even when the presence of infection is diagnosed, the patient may not look appreciably ill, and the treatment may therefore be inadequate Protracted infections, masked by inappropriate antibiotic therapy, are especially dangerous because a patient may be harboring a serious infection despite a normal temperature and a normal white blood cell count

Although septic shock is not commonly seen by the practicing physician, one must be alert for it when a pregnant patient has a gram negative infection Appropriate measures must be taken for its early recognition and treatment (see Ch 3, Shock) Several specific conditions that confront the obstetrician frequently in his practice and may progress to severe sepsis will be discussed

SEPTIC ABORTION

Elective abortion has become an accepted procedure in the United States since the Supreme Court decision of January 22, 1973 Whether this has markedly decreased the number of poorly executed illegal abortions, as is frequently claimed, or whether even these are being done more professionally, there is no doubt that fewer patients with infected abortions are now being seen However, sepsis following first- and second-trimester abortions has not been eliminated, and it remains as a significant cause of maternal morbidity and mortality

Diagnosis

The patient commonly presents with the characteristic signs of sepsis a temperature range of 100–105° F (38–40 6° C), lower abdominal pain or cramping, malaise, headache, and a serosanguineous discharge Frequently, because the patient thinks the symptoms are caused by the abortion, she delays seeking medical care Delay may also occur if she had received prophylactic antibiotics when the abortion was performed, as the infection may manifest itself only after cessation of therapy

Management

The following steps should be taken

1. Obtain upright and flat x ray films of the abdomen to look for interstitial gas, air under the diaphragm or the presence of a foreign body, so that perforation of the uterus may be ruled out
2. Obtain a chest film to rule out pneumonitis and to use as a base line for future reference
3. Draw blood for typing and crossmatching, complete blood count, serum electrolytes, blood urea nitrogen, serum creatinine, and sedimentation rate
4. Start an intravenous infusion of 5% dextrose with saline so that constant intravenous access is available

5. Insert a Foley catheter so that urinary output may be recorded hourly
6. Have vital signs recorded every 15 min until the patient has responded to therapy
7. Insert a central venous pressure catheter into the antecubital, subclavian, or external jugular vein and advance into the superior vena cava to record central venous pressure every 15 min or as indicated (normal range, 8–16 cm H O) Alternatively, insert a flotation catheter of the Swan Ganz type into the antecubital vein to measure the pulmonary artery wedge pressure (normal range, 6–12 mm Hg)
8. Obtain swabs of the endometrial cavity for Gram stained smear and for aerobic and anaerobic cultures
9. Administer antibiotics of the appropriate type intravenously, and preferably in combination Ampicillin (1–2 g every 4 hours) with gentamycin (1 3 mg/kg body weight every 8 hours) and clindamycin (600 mg every 6 hours) or chloramphenicol (1 g every 6 hours) are effective against almost all organisms
10. Undertake surgery to remove the septic focus within 6 hours Usually sharp curettage removes residual products of conception more effectively than blunt or aspiration curettage

In a patient who does not improve despite adequate medical treatment and curettage or who shows evidence of myometritis with peritonitis or who has a uterine perforation, a hysterectomy is usually necessary Most would agree that a hysterectomy is indicated when the abortion has been chemically induced

When gas gangrene is present, the uterus is usually very tender, and gas formation hemolysis, shock, and renal failure may occur A Gram stained smear is very useful for making an early diagnosis Intravenous crystalline penicillin supportive therapy, and hysterectomy give the best results Hyperbaric oxygen may be useful but polyvalent antiserum is not

ANTEPARTUM SEPSIS AND THE INTRAUTERINE DEVICE

Genital sepsis as a complication following the insertion of an IUD or related to an *in situ* device has been a recognized hazard for many years However, not until recently has the literature clearly described a symptom complex of overwhelming sepsis and maternal death caused by the presence of an IUD in the gravid uterus This problem usually develops late in the first semester or in the second trimester of pregnancy

Diagnosis

The patient often presents with symptoms suggestive of an upper respiratory tract infection In the presence of mild or moderate infection, abdominal discomfort and a brownish watery discharge are common In severely infected patients high fever, chills prostration, signs of septic shock, and death within 24–48 hours after the onset of symptoms have been reported

Management

If, upon speculum examination, the IUD string is visible, the IUD is best removed If the string is not visible, the patient must be treated as for a septic abortion with intravenous antibiotics intravenously, and uterine evacuation.

ACUTE PYELONEPHRITIS IN PREGNANCY

Pyelonephritis in pregnancy has been recognized as a major problem for over a hundred years Although it is a common cause of maternal morbidity, it is rarely a cause of maternal mortality

Kass has suggested that the patient with asymptomatic bacteriuria at the time of her first prenatal visit is at risk of developing pyelonephritis during her pregnancy The incidence of bacteriuria at the first prenatal visit has been reported to be approximately 6% of patients, and of these, approximately 30% develop pyelonephritis during the pregnancy More significantly, almost 30% of patients in whom bacteriuria was diagnosed during pregnancy were still bacteriuric 10–14 years later, and many showed signs or symptoms of chronic renal disease Pyelonephritis during pregnancy has also been reported to increase the incidence of premature delivery, but there is some disagreement about this However, the problem for the mother is a major one, and only with the prompt treatment of asymptomatic bacteriuria and the more adequate treatment of acute pyelonephritis will it be significantly diminished

Management

Each patient must be investigated for asymptomatic bacteriuria at the time of her first prenatal visit If bacteria are seen in a Gram stained smear from an unspun, clean catch urine sample or if a quantitative urine culture has a colony count in excess of 10^5/ml, the patient should be treated

Repeated evaluations throughout pregnancy and at the postpartum visit are essential If the patient develops symptoms of acute pyelonephritis during pregnancy, she should be admitted to the hospital at once and a Gram stained smear and urine culture (with antibiotic sensitivity testing) should be performed on a clean catch midstream or catheter specimen of urine A regular urinalysis should also be performed, of course A patient who is febrile, with or without costovertebral angle tenderness should be admitted to the hospital. Either ampicillin (1 g every 4 hours) or cephalothin (2 g every 6 hours), given intravenously, is usually effective, both drugs are relatively safe for use in pregnancy and are cleared via the urinary tract.

Occasionally, a patient presents with symptoms of pyelonephritis, but microscopic examination of the urine fails to reveal pyuria When this occurs, the physician must continue to observe the patient, and if pyelonephritis is still a major diagnostic consideration, a ureteral catheter must be passed to rule out obstruction Patients should be encouraged to lie on the left side because this allows better drainage of the right kidney during pregnancy

Antibiotic therapy should be continued for at least 7–10 days after remission of signs and symptoms of infection Some investigators, such as Kass (1970), suggest continuance of therapy for the duration of pregnancy

Close follow up of the patient after discharge is important because recurrence of infection is common Thorough evaluation of the urinary tract, including intravenous urography, is essential 4–6 weeks after delivery

Because of the prevalence of gram negative bacteria, especially *Escherichia coli* as the etiologic agents of pyelonephritis in pregnancy, the physician must be prepared to diagnose and manage the septic shock syndrome in these patients Although this is an uncommon cause of septic shock, management is difficult

because it is entirely medical, there being no readily removable septic focus involved

MATERNAL PNEUMONIA IN PREGNANCY

There are changes in every maternal physiological system during pregnancy Many of these are compensatory and protective in nature, and certain ones may explain the propensity of pregnant women to develop certain types of infection (urinary tract changes are the classic example) Changes in pulmonary physiology during pregnancy have been proposed as the basic explanation for the deadly qualities of a number of pneumonic processes in pregnancy From the health care viewpoint, it is essential not to underestimate the potential for danger that exists in upper respiratory infections during pregnancy Besides contributing to maternal mortality and morbidity, they may affect the fetus unfavorably Moreover, diagnostic and therapeutic measures that would ordinarily be utilized can affect the fetus Consequently, a complacent attitude on the part of the physician toward these conditions entails considerable risk, even though fatal complications are relatively rare

From the clinical standpoint, three of the more important pneumonias are 1) influenza pneumonia, 2) varicella zoster pneumonia, and 3) *Diplococcus pneumoniae* pneumonia Schwarz and Fruiterman have provided a succinct review of the subject (1976)

Influenza Pneumonia

Respiratory involvement may be caused by pure influenza virus pneumonia and a superimposed bacterial pneumonia In the pregnant woman, the viral form is thought to be more lethal, but differentiation is difficult and clinical management is the same for both Usually there are prodromal symptoms, lasting 6–12 hours They include malaise, myalgia, and chills followed by fever, headaches, eye pain, nasal congestion, or mild pharyngitis About the third day, signs and symptoms of pneumonia usually appear—cough, dyspnea, mucopurulent sputum or hemoptysis, and pleuritic chest pain Physical examination reveals basilar inspiratory rales, and the patient is acutely ill In severe cases, the chest x-ray film shows bronchopulmonic infiltration, frequently involving more than one pulmonary segment The WBC count may be low, normal, or moderately elevated Gram-stained sputum smears show an insignificant number of bacteria, unless superimposed infection has occurred

MANAGEMENT The majority of viral infections are relatively mild, but infection can be severe enough to cause maternal death, usually within 24–48 hours after the clinical features of pneumonia appear Severe cases require hospitalization, and reverse isolation is advisable Consultation with the pulmonary and anethesia services is advisable because of the likelihood of cardiopulmonary failure and the need for intubation Patient monitoring should include central venous pressure and arterial blood gas determinations Vigorous respiratory therapy is appropriate Antibiotic therapy is used because of the likelihood of superimposed bacterial infection, but only after sputum and blood cultures are obtained Oxacillin and gentamycin are currently the drugs of choice

Pregnant women are susceptible to influenza, and are therefore considered suitable candidates for influenza shots, especially when there is likelihood of an epidemic The viruses readily undergo mutation, however, and it is advisable to check the periodic public health bulletins on the subject The recommendations contained within them should be followed

Varicella-Zoster Pneumonia

This is the result of disseminated disease Clinical symptoms of pneumonia usually develop 1–6 days after the appearance of the skin rash The first signs are a dry, nonproductive cough with tachypnea, dyspnea, and a temperature of 102° F or more The cough may become productive and is often blood streaked. Chest x-ray examination may show marked vascular changes with characteristic acinonodular infiltrates

MANAGEMENT In the acute situation, treatment is similar to that used for influenza pneumonia Susceptible pregnant women may benefit from the administration of hyperimmune serum within 72 hours after exposure

Diplococcus Pneumoniae Pneumonia

In pregnancy, lobar pneumonia results in a 2–3% mortality Early recognition and aggressive therapy may be life saving Typically, onset is sudden, with a shaking chill, headache, sharp pain in the involved hemithorax, and cough with the early production of pinkish sputum Rusty sputum, when present, is practically diagnostic Dyspnea is marked The classic signs of consolidation may be lacking, but fine rales and suppressed breath sounds can be heard over the involved area Chest x-rays may yield the only positive evidence of pulmonary consolidation Sputum smears may show gram positive encapsulated diplococci and numerous polymorphonuclear leukocytes

MANAGEMENT Supportive therapy includes hydration and oxygen administration Antibiotic therapy is initiated after sputum and blood cultures are obtained Penicillin G, in intravenous doses of 20 million units per 24 hours, is recommended For patients allergic to penicillin, other drugs must, of course, be used —*e g* , erythromycin or lincomycin Cephalosporins are also effective but cross-reactions may occur Treatment should be continued until the patient has been afebrile for 48 hours

Relation to Labor and Delivery

These infections may occur at any time during pregnancy Moreover, they may initiate labor Although primary attention should be focused on the acute process, appropriate planning for the management of labor and delivery and for care of the newborn is also essential A recurring problem is the patient who is scheduled to have a repeat cesarean section, who then develops an acute respiratory problem 1 or 2 days before the elective operation In the interest of mother and infant, extreme caution is advisable The operation may have to be postponed

FETAL INFECTIONS IN PREGNANCY

Maternal infection may affect the fetus either by hematogenous spread across the placenta or by ascending infection from the maternal genital tract Many organisms, bacterial, viral, and protozoal can affect the fetus (Table 4-1) Our concern here, however, is with late perinatal infection or amniotic infection, commonly associated with premature rupture of the membranes and prolonged labor.

The incidence of neonatal infection following membrane rupture rises directly with the length of time from membrane rupture to delivery, and shows an inverse relationship with birth weight (Fig 4-1, 4-2) The work of Larsen, Snyder, and Galask (1974) demonstrates that amniotic fluid has bacteria-inhibiting factors and that this antibacterial effect is less effective in prematurity and postmaturity This finding explains at least in part, why some patients and not others develop amniotic fluid infection following membrane rupture When infection of the amniotic sac does occur, the fetus can swallow and inhale the infected material, and it may spread farther, to the bloodstream or meninges

The diagnosis of fetal infection is very difficult Fetal tachycardia is the only sign available to the obstetrician Since concomitant maternal infection will often be present, this is not very significant except as a sign of fetal distress, the treatment of which is the same as the treatment of maternal intrapartum infection—*i e*, delivery Cultures should be taken from every potentially infected infant, and the pediatrician must be informed of the possibility of neonatal pneumonia and septicemia

Viral Infections

Consideration of the various viral infections that can occur during pregnancy reveals that the gravida is often spared while the sequelae of these diseases impinge upon the fetus Recently, a group of several diseases has been labeled the **TORCH** complex The mnemonic TORCH is defined as follows **TO** for toxoplasma, **R** for rubella, **C** for cytomegalovirus, and **H** for herpes simplex The infectious agents of these diseases have little in common except their malign effects upon the fetus Toxoplasmosis is a parasitic disease which may cause serious disease in the fetus Rubella virus and cytomegalovirus cause congenital abnormalities Although herpes has not been shown to cause anomalies, it does cause severe infection and often the death of the baby Cytomegalovirus and herpes simplex are DNA viruses, while the rubella virus is an RNA virus

Route of Infection The fetus may become infected by several routes transplacental passage, direct invasion through the membranes, or direct contact during delivery (After birth, nursing infants may become infected by ingestion of infected milk) Examples of all of the routes have been documented except for direct invasion through the fetal membranes Since it has been shown that bacteria from the cervix or vagina can be isolated from the amniotic fluid after traversing the membranes, it is not unreasonable to assume that viruses can follow the same route

The obstetrician must be aware of the various disease entities and, whenever possible, be able to recognize them during the antepartum and early postpartum period Maternal antibody titers are valuable if acute and convalescent blood

TABLE 4-1. Fetomaternal Infection

Disease	Time of Transmission	Teratogenic Effects
Rubella	First trimester; early second trimester (problematical)	Cataracts glaucoma retinopathy, microphthalmia, microcephaly, congenital heart disease
Toxoplasmosis	First trimester	Microcephaly, hydrocephaly, cerebral calcifications chorioretinitis
Cytomegalovirus	Throughout pregnancy	Microcephaly, cerebral calcifications
Herpes simplex	First trimester (transplacental), intrapartum (transplacental), ascending and direct contact	Chorioretinitis microcephaly microphthalmia
Varicella	First trimester and intrapartum	Chorioretinitis microcephaly limb deformities
Group B coxsackie virus	First trimester and late in pregnancy	Provisional assn GU anomalies hare lip, cleft palate, congenital heart disease CNS anomalies, pyloric stenosis
Syphilis	Second half of pregnancy	Delayed effects on eyes ears teeth, joints CNS
Listeria	Intrapartum	Not known
Gonococcus	*Last trimester and at delivery*	*Not known*
Tuberculosis	Rarely transplacental usually following delivery	Not known

(Evans HE, Glass L Perinatal Medicine New York, Harper & Row, 1976)

Other Manifestations	Laboratory Diagnosis
Thrombocytopenia, hepatosplenomegaly, hepatitis, pneumonia, bone destruction, encephalitis, growth retardation	HI antibody, viral isolation from body fluids
Encephalitis, myocarditis, hepatosplenomegaly, jaundice, diarrhea, vomiting convulsions	CF, HI antibody, IgM-FTA specific antibody
Same as toxoplasmosis	Neutralizing or CF antibody, isolation of virus from urine inclusion-bearing cells in urine
Cutaneous lesions (vesicles), visceral involvement (granulomas)	Neutralizing or CF antibody, isolation of virus from chorioallantoic membrane tissue culture, growth of virus on rabbit cornea
Skin lesions, encephalomyocarditis, visceral involvement	Growth of virus on tissue culture, CF antibody
Encephalomyocarditis, pneumonia	Neutralization and CF antibody, growth of virus on tissue culture
Osteochondritis, jaundice, hepatosplenomegaly, lymphadenopathy, rhagades, anemia	Darkfield exam for spirochetes, FTA-ABS, TPI immobilization
epsis, meningitis, hepatitis, diffuse granulomatosis	Isolation of bacteria from blood, urine or pus
iepsis conjunctivitis, panophthalmitis	Gram stain and culture (Thayer-Martin medium)
Fever, anemia, pulmonary and systemic dissemination	Isolation of organism from gastric washing or maternal lesions, PPD unreliable during neonatal period

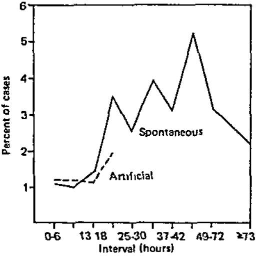

FIG 4-1 Relationship between definite infection in newborn infants and interval from membrane rupture to birth Solid line, Spontaneous rupture Dashed line, Artificial rupture Obstet Gynecol 28 22, 1966

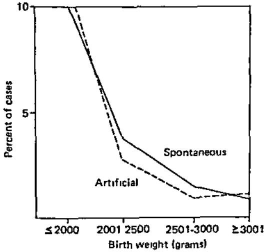

FIG 4-2 Inverse relationship between birth weight and definite infection in newborns (Shubek F, Benson RC, Clark WW, et al Fetal hazard after rupture of the membranes Obstet Gynecol 28 22 1966)

samples are collected during both the acute and convalescent stages of illness and are compared When an elevated titer is obtained, an attempt to isolate the virus or parasite from amniotic fluid should be made Umbilical cord blood should be collected for an immunoglobulin M (IgM) titer, which, when elevated, is highly suggestive of disease The pediatrician responsible for the baby should attempt to isolate the virus or parasite from the involved infant in order to confirm the diagnosis as early as possible in the neonatal period

HERPESVIRUS HOMINIS INFECTION DURING PREGNANCY This condition is not usually serious for the mother but deserves special mention because of its high mortality in the fetus and newborn Neonatal infection in the immediate postdelivery period results in fulminating disease with a mortality rate of over 50% Moreover, of the babies who survive, over 50% will manifest some form of neurologic or ophthalmic disease

TABLE 4-2. Clinical Differences Between Primary and Recurrent Vulvovaginitis Due to *Herpesvirus hominis* Type II

Signs or symptoms	Primary	Recurrent
Ulcerated lesions	Multiple	Scattered 1 to 3
Location of lesion	Tends to involve both labia and vagina cervix may be concomitantly involved	Limited involvement of vulva vagina or cervix
Size of lesions	Variable will be larger than those observed in recurrent disease	Tends to be smaller
Inguinal adenopathy	Present	Absent
Viremia	Occurs	Absent
Systemic symptoms (malaise myalgia fever)	Present*	Absent
Local symptoms (dysuria itching dyspareunia)	Present	Present
Specific antibody titer	Greater than fourfold rise observed between the pre- and postconvalescent serums	No significant change

*Only in the absence of pre-existing antibodies to Herpesvirus hominis type I

(Monif GRG Infectious Diseases in Obstetrics and Gynecology New York Harper & Row 1974)

When the condition is suspected during pregnancy, the obstetrician must decide on whether the attack is primary or resurgent Clinical differences between primary and recurrent infection are presented in Table 4-2 When it is not possible to differentiate by history and physical examination, several laboratory methods are available for this purpose

1. Serologic analysis, using complement fixation in testing for specific antibody to herpesvirus, appears to be the best indicator of primary infection When lesions first appear a blood specimen should be drawn, and a second specimen should be obtained 10 days later The presence of a significant antibody titer in the initial specimen and the failure of the titer to exhibit a fourfold increase in the second specimen strongly suggests recurrent infection The prognosis for the fetus is good Primary infection with either herpesvirus type I or type II, in the absence of cross protecting antibodies, exposes the fetus to the risk of congenital infection
2. A Papanicolaou smear of a herpetic lesion may demonstrate characteristic intranuclear inclusion bodies in multinucleated giant cells
3. Biopsy of a typical lesion may give the diagnosis
4. Viral cultures may be positive from a lesion or from the amniotic fluid (If the virus is present in amniotic fluid, then the fetus is almost certainly involved, and cesarean section will *not* be indicated unless for obstetric indications)

Management This has been succinctly summarized by Monif (Table 4-3) If lesions are present in the birth canal, the neonate usually becomes infected during passage through the canal It is possible, however, that transplacental trans-

TABLE 4-3. Clinical Management of Herpetic Vulvovaginitis in Pregnancy (Herpesvirus hominis Type II)*

Primary					Recurrent	
+ Cross-reacting antibodies to Herpesvirus hominis type I		− Cross-reacting antibodies to Herpesvirus hominis type I (Amniocentesis)				
		+ Herpesvirus	− Herpesvirus		Lesion at parturition	No lesion at parturition
Lesions at parturition	No lesions at parturition		Lesions at parturition	No lesions at parturition†		
Cesarean section	Vaginal delivery	Vaginal delivery	Cesarean section	Vaginal delivery	Cesarean section	Vaginal delivery

*Prior to 37th week of gestation

†If after 37th week of gestation herpesvirus cultures are positive within 48 hours after inoculation, irrespective of the absence of specific lesion cesarean section is advocated

(Monif GRG Infectious Diseases in Obstetrics and Gynecology New York, Harper & Row, 1974)

mission of the virus may already have occurred Thus, if a patient has a demonstrable lesion on the vulva, vagina, or cervix, and there is no evidence of intrauterine infection, and if the membranes are intact or have been ruptured less than 4 hours, then delivery should be by cesarean section This gives a good chance of avoiding infection of the infant

Bacterial Infections

Bacterial infections of the fetus and neonate are part of the complex problem of perinatal wastage Predisposing maternal factors such as prolonged rupture of membranes, prolonged labor even with membranes intact, and multiple vaginal examinations are well recognized The portal of entry is usually bacterial ascent from the vagina through the cervix, resulting in fetal and neonatal infection, significant causes of morbidity and mortality

Less well understood are the mechanisms that lead to fetal infection in the absence of the usual predisposing factors Evidence for such infection has been based on pathological evidence of pneumonic processes in the lungs of macerated and nonmacerated stillborn infants The proposed explanation is aspiration by inhalation and swallowing of infected amniotic fluid This is understandable only if it occurs while the fetus is alive and before it is born Blanc has proposed that hematogenous transmission is largely responsible for infection that occurs before the membranes rupture The possibility of bacterial ascent through the closed cervix is not excluded The relationship of this entity—amniotic fluid infection syndrome—to perinatal wastage has been stressed by Naeye and Blanc Supportive evidence has been provided by the demonstration of potentially pathogenic bacteria in amniotic fluid even in the presence of intact membranes Evidence for bacterial inhibition by amniotic fluid has also been developed According to Schlievert *et al* a phosphate sensitive bacterial inhibitor substance may be found in some amniotic fluids as early as the 20th week of gestation This inhibitory system is increasingly effective against gram negative bacteria as term approaches Whether this or a similar system provides protection against Group B streptococci is uncertain The fact that approximately only 6% of pregnant patients at term show the presence of this organism suggests similar protective physiological mechanisms

The concept of the amniotic fluid infection syndrome is still somewhat suspect from the clinical point of view At any time other than during labor, or after prolonged rupture of the membranes, recognition would be extremely difficult and treatment uncertain Occurrence of the syndrome in labor or after membrane rupture would be indicated by fetal tachycardia—a sign of fetal distress The treatment is delivery

Until concept of the amniotic fluid infection syndrome is fully developed, common sense suggests that physicians pay closer attention to the diagnosis and management of cervicitis and vaginitis during pregnancy Reconsideration of the advice given in regard to coital activity during pregnancy might be necessary The same applies to several obstetric practices, such as digital examinations during pregnancy or the casual stripping of the membranes as term approaches Lastly, nursery personnel should be alerted to the possibility of infection, particularly because of the difficulty in recognizing sepsis in the newborn Cultures should be taken from every potentially infected infant

INTRAPARTUM INFECTIONS

In the intrapartum period, chorioamnionitis (inflammation of the fetal membranes) is the most important type of infection It may result in life threatening infection for both mother and baby The threat is both immediate and remote, for the danger period extends into the puerperium for both Although infection such as that following pudendal block may occur, here the problem will be discussed mainly with regard to maternal mortality and serious morbidity

CHORIOAMNIONITIS

As pointed out by Webb in 1967, one maternal death occurred among 5500 patients with premature rupture of the membranes in the state of California The natural defenses against infection are the cervical mucus plug, the intact membranes, and antibacterial substances in the amniotic fluid

It is now well established that the incidence of chorioamnionitis is directly related to the duration of membrane rupture prior to delivery In 75% of patients with membranes ruptured for more than 24 hours, cultures obtained by amniocentesis are positive, even although only about 20% of these patients show any evidence of chorioamnionitis histologically following delivery Occasionally, however, overwhelming infection with gram negative aerobic bacilli (*E coli*) swamps the host's defenses, and septic shock develops When this occurs, it is much more difficult to manage than shock associated with abortion

Prevention

Prevention is obtained by optimal management of the asymptomatic patient with premature rupture of the membranes The following guidelines are reasonable, but much will depend on the type of patient population served

1. The patient should be admitted to the hospital for observation
2. A sterile vaginal examination should be carried out on admission if prolapsed cord is suspected and to assess the presentation, station, and degree of cervical dilatation should this complication be present
3. The following tests should be performed to determine definitively whether the membranes are ruptured
   - A. Vulvar observation for unmistakable evidence of copious amounts of amniotic fluid leakage
   - B. Nitrazine paper test (positive results due to alkaline amniotic fluid)
   - C. Fern test (positive if 1 drop of amniotic fluid allowed to dry shows fern ing)
   - D. Cytologic examination (positive if vernix caseosa cells are present)

The patient's history and these four tests will confirm the diagnosis with well over 90% accuracy

In the asymptomatic patient with a baby of less than 34 weeks' gestation and no underlying obstetric complications, management after diagnostic maneuvers is all that is indicated No prophylactic antibiotics should be used, because they may mask a low grade infection, but they should be given when labor starts There is some evidence that antibiotics reduce maternal morbidity in the afebrile patient who goes into labor, but there may be an increase in *Candida albicans*

infections seen in the newborn of these mothers At less than 34 weeks' gestation, the chance that a patient will fail to go into labor during 72 hours following rupture of the membranes is about 20%, as compared to about 10% after 34 weeks

If the patient develops overt signs of infection—*i e*, fever, uterine rigidity and tenderness, or a purulent vaginal discharge—labor should be induced at once. An oxytocin infusion should be started at 4 mU/min and the dose doubled every half hour until adequate contractions are achieved Antibiotics should be given as for septic abortion Vaginal delivery should be the aim, but if the membranes have been ruptured over 12 hours and delivery has not been achieved, then cesarean section should be considered

If at cesarean section the uterus is grossly infected, cesarean hysterectomy is the treatment of choice, because severe postpartum infection is practically inevitable Such complications as disseminated intravascular coagulation and septic shock may occur These problems have already been dealt with in detail in Chapter 3, Shock

In the asymptomatic patient with a gestation of more than 34 weeks, labor should be induced We have lost 3 of 7 patients with chorioamnionitis, as compared with 4 of 54 who had septic shock from other causes, and we feel that theoretic benefits to the baby, such as improved lung maturation, are not justification for procrastination

POSTPARTUM INFECTIONS

The main life threatening infections in this period—endometritis and septic pelvic thrombophlebitis—often derive from intrapartum or even antepartum chorioamnionitis

POSTPARTUM ENDOMETRITIS

Because this condition is common, some obstetricians have tended to become complacent and casual about it, and the results of treatment have consequently been poor However, more attention is once again being given to puerperal sepsis because of the realization that anaerobic bacteria and group B betahemolytic streptococci may be involved in both maternal and neonatal infection Also, puerperal sepsis may indeed be caused by a mixed flora (a fact that helps to explain some of the treatment failures) It is now better appreciated that Gram stained smears are useful, and that it is important to obtain adequate specimens for aerobic and anaerobic cultures The latter must be transported promptly to the laboratory and handled appropriately The choice of antibiotic should be based also upon recent hospital antibiotic sensitivity patterns, and adequate dosages should be used intravenously If a treatment failure does occur, the best choice of antibiotic can be made on the basis of the sensitivity patterns obtained from the previous culture The patient should then be reexamined and another culture specimen obtained before the antibiotic regimen is altered A search for abscess formation or pyometra should be performed at regular intervals until one is found and drained or until the patient responds to medical treatment Antibiotic administration should be continued for at least 72 hours after fever has subsided, to be certain of preventing recrudescence

In 1973, Sweet and Ledger reported on a 2-year experience with infectious puerperal morbidity. They found a 3.8% incidence of postpartum endometritis among 2691 deliveries. In spontaneous delivery or in operative trauma, commensal organisms as well as pathogens may give rise to infection in the uterus.

Endometritis is suggested by the presence of an otherwise unexplained fever in a postpartum patient who has a tender uterus and lower abdominal rebound tenderness. Mixed infections are now known to be the rule. Sweet and Ledger recovered (in the order of frequency) *Escherichia coli*, Peptostreptococcus, *Streptococcus viridans*, Bacteroides species, and enterococcus. Gibbs *et al.* found, in decreasing order of frequency, Peptostreptococcus, beta and alpha streptococcus, and *E. coli*. Their study often identified two or more organisms. It is of interest that a similar distribution of organisms was found in afebrile control patients. Many of the apparent discrepancies in reported microbiologic data are attributable to technique, both in obtaining and handling cultures. Vaginal and cervical contamination is a problem in obtaining endometrial cultures, and the way in which the material obtained is handled may dramatically affect the success in isolating anaerobes. So great care must be taken in the microbiologic evaluation of the patient with postpartum endometritis.

Management

The majority of responsible organisms will usually be sensitive to a combination of ampicillin (1–2 g every 4 hours) and gentamycin (1.3 mg/kg of body weight every 8 hours) given intravenously. Occasionally, for resistant *Bacteroides fragilis*, there will arise a need for clindamycin (600 mg) or chloramphenicol (1 g every 6 hours) intravenously. Because of the potential toxicity of the latter two drugs, they should not be utilized unless there is cultural or clinical evidence pointing to *Bacteroides fragilis* infection or unless septic shock is present. Failure of response to adequate antibiotic therapy suggests the existence of either a purulent collection requiring drainage or a pelvic thrombophlebitis. Occasionally it will be necessary to resort to examination under anesthesia or even to perform a laparotomy to reveal an abscess. If clinical findings are minimal and there is no leukocytosis, the possibility of drug fever should be considered. Although antibiotics are notorious for causing drug fever, other possibilities should be excluded first.

Most investigators recommend the use of ergot preparations to stimulate uterine contractions as an adjunct to antibiotic therapy. Easy passage of a large uterine sound should be made to eliminate the possibility of lochial obstruction, and dilatation and curettage must be considered if there is any question of retained placental fragments.

The incidence of morbidity from puerperal endometritis is even higher with cesarean section than with vaginal delivery. Sweet *et al.* reported rates of 13–27%. This has led a number of workers to consider the prophylactic use of antibiotics in patients having a cesarean section. Although such usage is still controversial, Gibbs *et al.* reported a significant reduction in puerperal morbidity from endometritis by means of a short perioperative course of antibiotics. Moreover, despite some views to the contrary, they observed no increased incidence of post cesarean infection in patients who had internal monitoring following ruptured membranes.

SEPTIC PELVIC THROMBOPHLEBITIS

As *Schwarz and Fruiterman (1976)* have pointed out, there are three logical therapeutic alternatives in the management of the patient with severe puerperal sepsis who is not responding satisfactory A review of the available bacteriologic findings may suggest the first alternative—a change in the antibiotics being used This is often desirable when certain gram negative rods or anaerobes are present

A careful pelvic examination may reveal the presence of a pelvic mass If this mass is an abscess, the second alternative may be necessary—surgical drainage by the abdominal or vaginal route

Finally, if there is no pelvic abscess and antibiotic therapy is appropriate, it is likely that there is septic pelvic thrombophlebitis (SPT) Current diagnosis and management of this entity reflect the considerable controversy surrounding it

Although the problem of puerperal sepsis and the life threatening complication of SPT have been appreciated for some time, SPT was not systematically investigated until 1951, when *Collins et al* reported on 70 patients in whom SPT was proved at laparotomy Between 1937 and 1946, they found that 35% of the patients who died of puerperal sepsis had evidence of septic thrombophlebitis The *National Center for Health Statistics in 1973* reported that 7% of patients who died of sepsis had SPT as the ultimate cause of death This improvement may be the result of better medical and surgical management of the primary pelvic infection rather than improved treatment of the septic pelvic thrombophlebitis

Diagnosis

When a patient becomes febrile during the immediate postpartum period and shows no evidence of infection elsewhere postpartum endometritis must be suspected Usually the uterus is subinvoluted and tender, and lower abdominal rebound tenderness is often present Intrauterine swabs should be obtained for Gram stained smears and for aerobic and anaerobic cultures, and blood should be taken for cultures Pending the reports on uterine and blood cultures, therapeutic antibiotics are selected on the basis of Gram stained smears If the patient does not respond within 72 hours to apparently appropriate antibiotics, if causes of fever other than endometritis have been excluded, and if there is no evidence of a pelvic abscess SPT must be strongly suspected

Abdominal pain and tenderness along the course of the hypogastric and/or ovarian veins may be present, as well as a propensity to shaking chills and tachycardia The presence of chest pain or bloody sputum suggests that pulmonary embolization has already taken place Confirmation of this diagnosis may be made by obtaining an isotopic lung scan or chest x-ray films (posteroanterior and lateral) In the pathogenesis of SPT, Bacteroides organisms play a prominent role, probably because of the production of heparinase by these organisms, and this should be kept in mind when antibiotics are chosen

Management

For patients with localized SPT, Schwarz and Fruiterman (1976) have suggested that the initial treatment should be anticoagulation therapy with heparin. They believe that the agent should be given intravenously every 4 hours, in a

dosage of 5000–10,000 units and should be monitored by determination of the partial thromboplastin time The patient who has had full antibiotic therapy and continues to have the characteristic fever curve is considered to be the ideal candidate for a therapeutic test of heparin (Obviously those patients with meta static abscesses are excluded, as are those with evidence of vasomotor instability and single pulmonary embolus or multiple emboli in spite of adequate heparin therapy)

Schwarz and Fruiterman believe that an adequate clinical trial of antibiotics for 48–72 hours (if there is no rapid deterioration) can serve not only as good therapy but also as a useful diagnostic test Many patients with a presumptive diagnosis of SPT obtain defervescence from heparin and antibiotic therapy However, laparotomy should be carried out in those who show no evidence of response, as pelvic abscess requiring drainage will be found in many of the patients with suppurative thrombophlebitis

We feel that if a definite diagnosis of SPT is made, surgery is the treatment of choice, because these patients die of coalescing lung abscesses rather than from pulmonary infarction All are agreed that surgical therapy has a major role for those patients who do not respond to anticoagulant and antibiotic therapy, who have recurrent pulmonary emboli despite anticoagulant therapy, and for whom anticoagulants are contraindicated The procedure of choice is ligation of the major venous return from the pelvis—*i e*, the ovarian veins and the inferior vena cava It may be necessary to excise the veins, if the amount and extent of suppuration are great Patients with myometritis require hysterectomy and bilateral salpingo-oophorectomy to remove all septic foci Surgical therapy does not provide full protection against recurrent thromboembolism and thus con tinued anticoagulation is recommended No effect on future childbearing has been seen in patients who have had pelvic venous ligation, and swelling of the legs is uncommon when the operation is performed on the postpartum or post abortal patient

Antibiotics and heparin should be continued for at least 10 days after diagnosis or laparotomy Prolonged anticoagulation is used for the patient with documented pulmonary emboli This has been advocated by Ledger and Peterson and is particularly important in cases involving anaerobes As Schwarz and Fruiterman have pointed out, further improvement in maternal mortality from SPT depends upon the prevention of puerperal sepsis, a high degree of suspicion in cases of resistant puerperal fever, and prompt initiation of appropriate therapy The only real disagreement at the present time is as to what constitutes appropriate therapy

ANAEROBIC INFECTIONS IN OBSTETRICS

These infections have been alluded to throughout this chapter, but in view of the increasing attention currently accorded them, the following points are worth emphasizing

1 Anaerobic infections are not new, but closer cooperation between the clinician and the microbiologist and better culture techniques have brought them into prominence
2. The infections in which they participate are generally mixed infections A facultative pathogen produces the initial infection with tissue necrosis encouraging secondary invasion by anaerobes The necrosis spreads con-

centrically, encouraging the development of abscesses that are frequently filled with foul smelling pus Gas formation with crepitation is not uncommon even in the absence of *Clostridium perfringens*

3. Abscess cavities must be drained adequately before the infection can be controlled
4. Although most anaerobes are susceptible to ampicillin, penicillin, and cephalosporins, the presence of Bacteroides species calls for the use of clindamycin or chloramphenicol
5. In view of growing awareness of the significance of anaerobic infections, follow up must continue for months after the start of treatment so that all late infections are detected In these days of frequent use of antibiotics plus early discharge from the hospital, many postpartum patients who are not febrile may be harboring serious infection As pointed out by Ledger and Nakamura, the accepted definition of postpartum febrile morbidity (an oral temperature 100 4° F or more on any two of the first ten postpartum days), has definite limitations

GONORRHEA IN PREGNANCY

Neisseria gonorrhoeae may on occasion be isolated from asymptomatic pregnant patients Activation of clinical disease gives rise to localized, uncomplicated gonorrhea, particularly in early pregnancy A disseminated form, characterized by dermatitis, arthritis, and carditis has also been recognized Diagnosis is difficult In either type a positive culture is considered essential to establish the diagnosis Recently a strain of *Neisseria gonorrhoeae* has been reported that is penicillin resistant because of the production of penicillin inactivating beta lactamase The fetus is unaffected by maternal gonorrhea except that the presence of Neisseria in the vaginal tract may lead to gonococcal ophthalmia or even arthritis and septicemia in the neonate

Treatment regimens are to be found in U S Public Health Service Bulletins

Treatment of Uncomplicated Gonorrhea in Pregnant Patients

DRUG REGIMEN OF CHOICE Aqueous procaine penicillin G (APPG), 4 8 million units intramuscularly, divided into at least two doses and injected at different sites at one visit, together with 1 g of probenecid, by mouth, 30 min before the injections

Alternative Regimens For patients in whom oral therapy is preferred, ampicillin, 3.5 g by mouth, together with 1 g probenecid by mouth, administered at the same time There is evidence that this regimen may be slightly less effective than the recommended APPG regimen

PREGNANT PATIENTS WHO ARE ALLERGIC TO PENICILLINS There are several possible alternative regimens, each of which has potential disadvantages

1. Erythromycin, 1 5 g orally, followed by 0 5 g four times a day for 4 days, for a total of 9 5 g This regimen is safe for mother and fetus but efficacy has not been established Erythromycin estolate should not be used in patients with liver disease

2. Spectinomycin, 4 g intramuscularly in two sites This is an effective dose, but safety for the fetus has not been established

CONTRAINDICATED DRUG Tetracycline should not be used for uncomplicated gonococcal infection in pregnancy because of potential toxic effects upon mother and fetus

Treatment of Disseminated Gonococcal Infection

Effective treatment schedules for the arthritis-dermatitis syndrome are as follows

1. Aqueous crystalline penicillin G, 10 million units intravenously per day for 3 days, or until there is significant clinical improvement This may be followed by ampicillin, 500 mg four times a day orally, to complete 7 days of antibiotic treatment
2. Ampicillin, 3 5 g orally, plus probenecid, 1 g, followed by ampicillin, 500 mg four times per day orally, for at least 7 days

In patients allergic to penicillin and/or probenecid, the following drugs may be substituted

1. Tetracycline, 1 5 g orally, followed by 500 mg four times a day orally, for at least 7 days, unless contraindicated
2. Erythromycin, 0 5 g intravenously every 6 hours, for at least 3 days

Additional measures are as follows

1. Hospitalization is indicated in patients who are unreliable, have an uncertain diagnosis, or have purulent joint effusions or other complications
2. Immobilization of the affected joint(s) appears helpful Repeated aspirations and saline irrigations appear beneficial, but controlled studies of these procedures have not been performed Open drainage of joints other than the hip is now generally discouraged in patients with gonococcal arthritis
3. Intraarticular administration of penicillin is unnecessary, since penicillin levels in the synovial fluid of inflamed joints approximate serum levels, furthermore, intraarticular injection *per se* may produce a toxic synovitis

Meningitis and endocarditis due to the gonococcus require high-dose intravenous penicillin therapy (at least 10 million units per day) for longer periods, usually at least 10 days for meningitis and 3–4 weeks for endocarditis

Prevention of Neonatal Infection

At the initial prenatal visit, all pregnant women should have endocervical cultures examined for gonococci as an integral part of prenatal care

PREVENTION OF GONOCOCCAL OPHTHALMIA

1. One drop of 1% silver nitrate solution applied to each eye at birth Do not irrigate with saline afterwards, for this may reduce efficacy
2. Ophthalmic ointments containing tetracycline, erythromycin, or neomycin are probably effective also
3. NOT RECOMMENDED Bacitracin ointment (not effective) and penicillin drops (sensitizing)

MANAGEMENT OF INFANTS BORN TO MOTHERS WITH GONOCOCCAL INFECTION Orogastric and rectal cultures should be taken from all such infants Blood

should be taken for cultures if septicemia is suspected If cultures or Gram-stained smears reveal gonococci, aqueous crystalline penicillin G, 50,000 units per kilogram of body weight per day, should be administered intravenously in two daily doses The duration of therapy should be determined by clinical response In suspected septicemia, an aminoglycoside should also be administered

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Acute Renal Failure

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Chapter 5

To produce - *Science begets knowledge opinion ignorance"*
Hippocrates

Acute renal failure is an uncommon complication of pregnancy, occurring in 1 1400 to 1 5000 pregnancies, depending upon the population being considered Nevertheless, it presents a serious problem and is being dealt with separately in this chapter because it has relevance to many conditions that will be considered in more detail later The importance of the subject comes into perspective when we consider that the mortality for acute renal failure ranges from 40–50% for acute tubular necrosis (ATN) to over 90% for acute cortical necrosis (ACN) The mortality can be reduced by the appropriate treatment of predisposing conditions, such as hemorrhagic shock, septic shock, abruptio placentae, and toxemia of pregnancy, and by the early use of dialysis when this is indicated

Acute renal failure is the sudden cessation of renal function, manifested by anuria, oliguria (less than 400 ml of urine per day) and increasing azotemia Clinically, according to the site of origin, it is useful to divide acute renal failure into three types 1) prerenal, 2) renal, and 3) postrenal The differentiation is sometimes difficult to establish, but early distinction between these three types helps to determine the proper therapeutic approach

CLASSIFICATION

Prerenal Failure

In obstetric patients with prerenal failure, the etiology of the renal insufficiency is usually dehydration or sodium depletion The condition is usually reversible if renal perfusion is improved promptly Usually, prerenal failure does not produce permanent changes in renal morphology However, if the condition producing prerenal failure is not promptly corrected, renal parenchymal failure can occur

Renal Failure

True renal parenchymal failure occurring during pregnancy is usually due to acute cortical necrosis or acute tubular necrosis Acute renal cortical necrosis is seen in patients who have suffered from prolonged and severe renal ischemia or in whom intravascular coagulation has occurred Morphologically, there is extensive destruction of glomeruli and tubules, with disappearance of cell nuclei and cellular boundaries, and obliteration of capillary and tubular lumens If the cortical necrosis is bilateral and total, this condition is irreversible However, in a few cases, the necrosis may be less severe and is patchy in distribution, a situation that accounts for the rare patient who survives with markedly impaired renal function In acute tubular necrosis, the blood vessels and glomeruli do not show significant morphologic changes but the tubules do The renal tubular epithelial cells may appear swollen or necrotic Some tubules are dilated, and the epithelium is flattened Granular or hyaline casts may be seen in the dilated tubules Examination of the urine may show renal tubular epithelial cells individually or in casts Interstitial edema and a nonspecific inflammatory cell infiltrate may be seen Acute tubular necrosis is usually reversible after an oliguric period lasting an average of 10–14 days

Postrenal Failure

In the condition of postrenal failure obstruction of the urinary tract beyond the glomerulus, as from a ligated ureter, results in an increased intraluminal pressure throughout the collecting system and causes a fall in the glomerular filtration rate Pyelonephritis frequently complicates the picture further increasing the parenchymal damage Microscopically, tubules are dilated and contain colloid casts There may be foci of interstitial inflammation throughout the medulla and cortex This condition is potentially reversible, but the prognosis varies depending on the duration and severity of obstruction before corrective treatment

ETIOLOGY

A discussion of all the causes of acute renal failure is beyond the scope of this chapter We shall consider only the syndromes commonly seen in the obstetric patient

Prerenal Failure

Prerenal failure is most often the result of sodium deficiency and extracellular volume depletion This condition may develop in patients who are on low sodium diets and/or receiving diuretics The patients will usually show clinical signs of dehydration A patient who is hypoalbuminemic may demonstrate peripheral edema, but because of intravascular volume depletion she may be in prerenal failure

Renal Failure

Acute tubular necrosis is the major cause of renal failure in association with pregnancy In abortion, chorioamnionitis, and pyelonephritis, endotoxic shock may cause it In some instances, the hemoglobinuria resulting from the hemolytic effect of clostridial infection may cause acute tubular necrosis

Nephrotoxic substances such as soap or Lysol used in illegal chemically induced abortions have been implicated The intravascular coagulation associated with intraamniotic hypertonic saline abortion has been shown to cause this syndrome Antibiotic therapy may also cause renal tubular damage Cephaloridine, amphotericin B, colistin, and the aminoglycosides (streptomycin, kanamycin and gentamycin) are the most frequent offenders The shock and hemoglobinuria resulting from a hemolytic transfusion reaction may result in acute tubular necrosis Hemorrhagic shock due to abruptio placentae, placenta previa, rupture of the uterus, postpartum uterine atony and operative trauma may also result in acute tubular necrosis

Bilateral cortical necrosis is rare By far the most common cause is abruptio placentae but the condition has also been reported with eclamptogenic toxemia, endotoxic shock and disseminated intravascular coagulation (DIC) Antecedent nephrosclerosis appears to increase the vulnerability of the kidney to this lesion As Chugh *et al* (1976) point out only mild DIC may be found in patients with acute cortical necrosis

The entity of postpartum acute renal failure has been described fairly recently In this syndrome, bilateral renal cortical necrosis and DIC occur within the first 6 weeks following an uncomplicated pregnancy and delivery The cause of the syndrome is obscure, but suggested factors include drug sensitivity (ergot), and a primary immunologic mechanism

Postrenal Failure

Postrenal azotemia in obstetric patients is most often due to obstructive uropathy, secondary to cervical malignancy, leiomyomas, and ovarian neoplasms complicating pregnancy If the patient has had a hysterectomy, a cesarean section, or a cesarean hysterectomy, the possibility of a retroperitoneal hematoma or ligated ureters must be considered

PREVENTION

From this brief discussion, it is clear that the occurrence of acute renal failure associated with pregnancy is often preventable An effective program of prevention should include several steps

1. Prompt replacement of blood in the event of massive hemorrhage
2. Careful observation for early signs of septic shock in patients with infected abortions, puerperal sepsis, chorioamnionitis, or pyelonephritis.
3. Early detection and prompt hysterectomy in uterine infections caused by *Clostridium welchii*, or when soap or Lysol have been used to induce an abortion
4. Avoidance of elective abortion induced by hypertonic saline injection
5. Careful monitoring of nephrotoxic drugs with frequent blood urea nitrogen and serum creatinine levels

6. Prompt detection and correction of intravascular volume depletion
7. Meticulous care to avoid the administration of incompatible blood
8. Early delivery in cases of abruptio placentae and severe eclamptogenic toxemia
9. Heparinization in selected cases of DIC, particularly when this is present after the uterus has been emptied or after hysterectomy
10. A pelvic examination with cytologic evaluation of the cervix at the first antenatal visit to rule out pelvic lesions that may cause obstructive uropathy

DIAGNOSIS

The diagnosis of acute renal failure is not difficult to make Frequently the earliest sign is a rising blood urea nitrogen, or serum creatinine Anuria or oliguria may be one of the presenting signs, but some patients go through the entire course of acute tubular necrosis without becoming oliguric (nonoliguric renal failure) Although sometimes difficult, it is essential that prerenal and postrenal failure be differentiated from renal parenchymal failure Prompt differentiation of these three conditions is necessary because their management is quite different Prerenal and postrenal failure are potentially reversible in their early stages but will progress to renal parenchymal failure if allowed to persist

PRERENAL FAILURE

In obstetric patients, the most common causes of oliguria are hypovolemia and hypotension producing prerenal failure by hypoperfusion On physical examination, a patient who is hypovolemic will show poor turgor of the skin, dry mucous membranes, and poor filling of the neck veins when in the supine position A low central venous pressure (CVP), 0–5 cm H O, serves to confirm the diagnosis of extracellular fluid volume depletion The CVP also helps in monitoring the replacement of the fluid deficit

The differentiation between prerenal failure and acute tubular necrosis may be aided by an analysis of the blood and urine chemistry (Table 5 1) These tests require only a random urine specimen of 5–10 ml and a 10-ml sample of blood The differential points are valid only if the kidneys were functionally normal before the acute insult and may not be seen if the patient has recently received a potent diuretic Moreover, congestive heart failure can produce pre renal failure with the same findings on examination of the urine as volume

TABLE 5-1 Differentiation of Acute Tubular Necrosis from Prerenal Azotemia

	Prerenal azotemia (mEq/liter)	Acute tubular necrosis (mEq/liter)
Urine sodium concentration	<20	>50
Urine creatinine/serum creatinine	>40	<10
BUN/serum creatinine	>20	<10
Urine osmolality/serum osmolality	>1 5	<1 1

depletion However, the differentiation between congestive heart failure and volume depletion can be made on the basis of physical findings

If the physical findings and the blood and urine chemistries suggest that hypovolemia is the cause of the oliguria, then a therapeutic trial of volume expansion for oliguria due to hypoperfusion is indicated

IMMEDIATE MANAGEMENT

In the absence of elevated CVP or signs of congestive heart failure,
Give normal saline, 500 ml IV in 30–45 min
Then give mannitol, 25 g IV, and
Furosemide, 120 mg IV
If urine flow increases to at least 40 ml/hr,
Replace the estimated extracellular fluid volume deficit with 0 9% NaCl and
Replace measured urine loss with 0 45% NaCl

FURTHER MANAGEMENT

If no response occurs in 2 hours and CVP is not elevated
Give normal saline, 500 ml IV in 45 min, and
Furosemide, 240 mg IV

For this therapeutic test to be effective the patient s blood pressure must be adequate to produce glomerular filtration (mean blood pressure at least 60 mm Hg), and the serum albumin concentration must be sufficient to maintain intravascular volume If there is no response after the second dose of saline and furosemide in the presence of normal blood pressure and CVP, the patient's oliguria more probably is due to acute tubular necrosis rather than prerenal failure

When osmotic agents such as mannitol are used the CVP, or alternatively the pulmonary capillary wedge pressure, as measured by a Swan Ganz catheter, should be monitored during the infusion The patient should also be examined repeatedly for signs of impending congestive heart failure, such as an increase in heart rate, a gallop rhythm, or the presence of basilar rales in the lungs If these occur, or if the CVP rises over 15 cm H_2O, or the pulmonary capillary wedge pressure over 12 mm Hg, then the infusion should be discontinued

POSTRENAL FAILURE

Visualization of an unobstructed renal collecting system will rule out postrenal failure If the patient is not anuric, and sufficient renal function remains, a high-dose intravenous urogram (IVP) may visualize the renal collecting system sufficiently to rule out obstruction If the renal function is too poor to visualize the collecting system by means of an IVP, cystoscopy and retrograde urography are indicated to rule out obstruction Note that for obstruction to produce renal failure, the outflow of both kidneys must be blocked, because the presence of one normal, unobstructed kidney is sufficient to prevent azotemia If an obstruction is found, a period of catheter drainage may be indicated to improve the patient's condition before definitive surgical correction of the obstruction is attempted

If DIC (synonymous with ICF) is suspected, a coagulation profile should be obtained (see Ch 2, Clotting Disorders in Pregnancy) and a smear of the

peripheral blood examined Thombocytopenia, the presence of schistocytes, prolonged prothrombin and partial thromboplastin times, decreased fibrinogen and elevated fibrin split products strongly suggest a consumption coagulopathy In some of these cases, the early removal of the source of procoagulant substances may save the kidneys Some of these patients may benefit from heparin therapy

RENAL CORTICAL NECROSIS

The diagnosis of renal cortical necrosis is difficult to establish The presence of red blood cells or red blood cell casts in the urine and the occurrence of anuria are seen more frequently with renal cortical necrosis than with acute tubular necrosis Renal biopsy showing necrosis of cortical glomeruli and destruction of other cortical structures establishes the diagnosis The appearance of a thin line of calcium, outlining the cortex of the kidney in a radiograph taken 4–6 weeks after the onset of renal failure, suggests the presence of renal cortical necrosis Patients with bilateral renal cortical necrosis seldom recover enough renal function to survive without dialysis These patients are usually candidates for chronic hemodialysis or renal transplantation

PATHOPHYSIOLOGY OF ACUTE RENAL FAILURE

Three pathogenic mechanisms have been postulated for the reduction in glomerular filtration rate and oliguria seen in acute tubular necrosis 1) Leakage of glomerular filtrate occurs through disrupted tubular basement membranes into the interstitial space From there it is reabsorbed, thus reducing urine volume and returning the filtered solutes to the circulation 2) Obstruction of the renal tubular lumen may result from sloughed tubular cells or swelling of the tubular epithelial cells and interstitial edema 3) Redistribution of intrarenal blood flow away from the outer cortical nephrons to the inner cortical and medullary regions results in a decrease in the glomerular filtration rate Differential intrarenal blood flow studies have established that this third mechanism of altered intrarenal blood flow is a factor in the acute tubular necrosis due to both ischemic and nephrotoxic lesions

Experimental evidence suggests that the renin angiotensin system may be the mediator of these hemodynamic changes, perhaps by the intrarenal conversion of angiotensin I to angiotensin II Hypotension associated with hemorrhage or infection is an important factor in the development of this condition In conditions such as abruptio placentae, septic shock, hemorrhagic shock and eclamptogenic toxemia, DIC may occur and the occlusion of glomerular capillaries by fibrin thrombi may result in acute tubular or acute cortical necrosis The pregnant state, with its relative hypercoagulability and depression of the fibrinolytic system, seems to predispose to intravascular coagulation Although acute cortical necrosis is more common in late pregnancy, it may also occur in early pregnancy

In most cases of acute renal failure, several of these mechanisms combine to reduce the renal blood flow, the glomerular filtration rate and the urine volume

BASIC MANAGEMENT OF ACUTE RENAL FAILURE

The clinical course of acute tubular necrosis is usually divided into three distinct stages the oliguric, the diuretic, and the recovery phase They are related in time to the initial insult and the process of repair

In the oliguric phase the urine output is usually less than 400 ml/24 hours This phase lasts from a few days to several weeks, and is characterized by increasing azotemia and hyperkalemia The excreted urine is dilute and has a specific gravity close to plasma ultrafiltrate (1 010)

The diuretic phase is manifested by an increasing and sometimes excessive urine output, which may reach 6–7 liters per day The urine is still dilute and renal function may remain severely impaired Azotemia is still present, and there may be marked electrolyte imbalance due to massive loss of electrolytes in the urine Almost 25% of the fatalities from acute tubular necrosis occur during the diuretic phase

The recovery phase is manifested by a return to normal urine volume and a gradually improving renal function Most patients reaching the recovery phase eventually regain nearly normal glomerular filtration rates

The mortality of acute tubular necrosis remains approximately 50% in spite of the general availability of dialysis Previously, the most common causes of death were azotemia pulmonary edema, or cardiac arrhythmias induced by hyperkalemia Today, most deaths associated with acute renal failure are caused by infection or by the primary disease The aim of therapy in acute tubular necrosis is to support the patient until renal function returns

The major areas to be considered in the management of acute renal failure are water balance, nutrition acid base balance, infection, cardiovascular complications, gastrointestinal complications, anemia, and coagulopathies

WATER BALANCE

Fluid intake in the afebrile patient should be limited to the volume of measured fluid output, such as urine or gastrointestinal drainage, plus 400 ml/day to replace insensible water loss If the patient is febrile, 100 ml of additional fluid is allowed for each 1° C temperature elevation Accurate recording of intake and output and daily measurement of the patient's weight are essential in managing water balance If fluid balance is being maintained properly, the patient should lose from 0 2 to 0 3 kg of weight per day If the patient's weight remains stable or increases, she is in positive fluid balance and fluid intake should be restricted further

NUTRITION

The basic principles of nutrition in acute renal failure are to minimize protein intake and to provide at least 100 g of carbohydrate per day to reduce the endogenous protein catabolism If the patient can tolerate oral feeding, it is best to give the carbohydrate by mouth since this allows a larger number of calories to be administered in the small volume of fluid the patient is allowed Several preparations are available commercially that provide high concentrations of glucose in a small volume of fluid A mixture of equal parts of Karo syrup

and ginger ale will also provide a high concentration of glucose and is well tolerated when chilled and flavored with lemon juice

If the patient cannot tolerate oral feedings, glucose must be administered intravenously This is best accomplished by the administration of 20–50% glucose through a central venous catheter, which has been passed into the vena cava It is recommended that 1000 units of aqueous heparin be added to each liter of intravenous fluid to prevent clotting of the venous catheter Because of the high incidence of septicemia associated with central venous catheters, scrupulous care must be taken in their insertion and maintenance Anabolic steroids, such as nandrolone decanoate (Deca Durabolin), may decrease the endogenous protein breakdown and slow the progression of azotemia Multi vitamins should be administered daily

ACID BASE AND ELECTROLYTE BALANCE

Although the balance of all extracellular electrolytes is disturbed to some degree in acute renal failure, the disturbances that require the most attention are acidosis and hyperkalemia Acute renal failure results in metabolic acidosis The course of the acidosis is best followed by monitoring arterial blood gases and serum bicarbonate levels Although the metabolic acidosis may cause tachypnea, it is usually well tolerated by these patients until the serum bicarbonate falls below 15 mEq/liter The acidosis is treated by the intravenous administration of sodium bicarbonate However, repeated injections of sodium bicarbonate may lead to overexpansion of the extracellular fluid volume as a result of the administration of large amounts of sodium For this reason, the management of persistent or severe metabolic acidosis usually requires dialysis

Hyperkalemia

Potassium excess is a common and serious problem in the management of acute renal failure and can cause cardiac arrest unless it is promptly diagnosed and treated All patients in acute renal failure are at risk of developing hyperkalemia, even if their potassium intake is restricted A catabolic state due to shock, trauma, sepsis, or an inadequate caloric intake can cause elevation of the serum potassium Acidosis causes hyperkalemia by producing a shift of potassium from the intracellular to the extracellular compartment Some frequently unrecognized sources of exogenous potassium are stored whole blood (10–30 mEq of K^+ per 1000 ml of serum) and potassium salts of penicillin (1 2 mEq of K^+ per million units of penicillin) The serum potassium concentration should not be allowed to rise above 6 mEq/liter because when the level reaches 7 mEq/liter, serious cardiac arrhythmias or asystole may occur Hyperkalemia may cause muscle weakness, decreased deep tendon reflexes, acro paresthesias, and an irregular pulse However, cardiac toxicity may occur before any symptoms of hyperkalemia are apparent Frequent electrocardiographic monitoring is essential in these patients The electrocardiographic abnormalities seen progressively with increasing hyperkalemia are tall, symmetrically peaked T waves, especially in leads V_3 to V_6, lengthening of the P R interval, widening of the QRS complex, disappearance of P waves and formation of a sine-wave configuration of the QRS complex (Fig 5-1) If the serum potassium con-

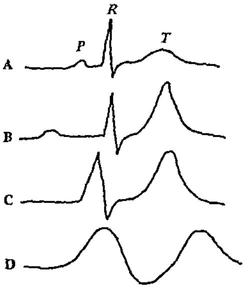

FIG 5 1 ECG changes of hyperkalemia. A Normal ECG B T wave 'tented and P–R interval lengthened C P wave disappears and QRS complex widens D 'Sine wave pattern

centration is 7 mEq/liter or higher, or if the electrocardiogram shows signs of hyperkalemia, one or more of the following measures should be taken immediately

1 Give 10 ml of 10% calcium gluconate intravenously over 3 min Calcium decreases the irritability of the cardiac muscle and thereby counteracts the effect of potassium on the heart Calcium salts should not be administered in the same container or through the same tubing as sodium bicarbonate because this would cause the precipitation of calcium carbonate Intravenous calcium should not be given to patients who are receiving digitalis, because this combination may produce cardiac arrhythmias
2. Give 1 ampule (44 6 mEq) of sodium bicarbonate intravenously over 5 min This may be repeated if necessary after rechecking the serum potassium concentration However, the danger of congestive heart failure from administration of the sodium should be kept in mind The relative alkalosis produced by the sodium bicarbonate drives potassium from the extracellular space back into the cells, thereby lowering the serum potassium level
3 Administer 300 ml of 20% glucose with 20 units of regular insulin at a rate of 100 ml/hour As the glucose is deposited in the cells in the form of glycogen, potassium also enters the cell This method will reduce the serum potassium level as long as the infusion is continued and perhaps for an hour after the infusion is stopped However, at that point potassium would again leave the cells and enter the extracellular space, causing a rebound hyperkalemia

All three of these acute measures for lowering the serum potassium have their onset of action within 5–15 min However, their effect rapidly disappears as soon as they are discontinued At the same time as these measures are being

employed, a cation exchange resin should be administered to decrease total body potassium, or dialysis should be initiated

Polystyrene sulfonate resin (Kayexalate) is a cation exchange resin that will bind potassium ions and remove them from the body It exchanges potassium ions for sodium ions on a one-to-one basis and therefore can be the source of a large sodium load Since its onset of action is about 1 hour after administration, Kayexalate is not suitable for rapidly lowering the serum potassium in patients with severe hyperkalemia It is usually given as a retention enema in a suspension containing 50 g of Kayexalate in 200 ml of 10% dextrose in water, or in 100 ml of 70% sorbitol and 100 ml of water The enema should be retained for 1 hour, and it may be repeated as often as necessary to lower the serum potassium concentration to the desired level Kayexalate may also be administered orally as 30 g (2 heaping teaspoonsful) suspended in a few ounces of water or ginger ale The administration of one ounce of 70% sorbitol at the same time facilitates transit of the resin through the gut and reduces the chance of impaction of the Kayexalate Each gram of Kayexalate will bind approximately 1 mEq of potassium In the adult patient, the average fall in potassium after a 50 g dose is about 1 mEq/liter After the severe hyperkalemia has been controlled, Kayexalate can be administered in doses of 20–50 g/day to prevent subsequent rise of serum potassium However the risk of vascular overload from the sodium administered (1 mEq sodium gained for each mEq potassium lost) must be considered

If the above measures do not reduce the toxic levels of potassium, then peritoneal dialysis or hemodialysis must be performed as an emergency measure to lower the serum potassium concentration

Hyponatremia

Although hyponatremia is often seen in acute renal failure, it is not an indication for the administration of sodium When it occurs in acute renal failure, hyponatremia almost always indicates an excess of water (dilutional hyponatremia) rather than a deficit of sodium Further restriction of water intake is then indicated

Hyperphosphatemia

The hyperphosphatemia which usually accompanies acute renal failure can be controlled by the oral administration of aluminum hydroxide gel, 30 ml, three times daily with meals

INFECTION

The most common cause of death in patients with acute renal failure is sepsis These patients are more susceptible to infection and once an infection becomes established, it is much more difficult to treat a patient with renal failure Furthermore, a diagnosis of infection may be difficult in these patients since the usual febrile response to infection may be suppressed in uremia

Prophylactic antibiotics should not be used However, as soon as the presence of an infection is established, treatment with appropriate antibiotics should be initiated The dose of most antibiotics must be reduced in acute renal failure,

TABLE 5-2. Modification of Antibiotic Dosage in Acute Renal Failure

Antibiotic	Nephrotoxicity	Average maintenance dose	Interval
Penicillins			
Ampicillin	No*	0.5 g	12 hr
Carbenicillin	No	2 0 g	12 hr
Methicillin	*No**	*1 0 g*	*8 hr*
Oxacillin	No	0 5 g	6 hr
Penicillin G	No	1 million units	12 hr
Cephalosporins			
Cephalexin	?	0.25 g	24 hr
Cephaloridine	*Yes*	*(Do not use)*	*(Do not use)*
Cephalothin	Probably	0 5 g	8 hr
Cephazolin	?	0 2 g	24 hr
Tetracyclines			
Tetracycline	Not†	(Do not use)	(Do not use)
Doxycycline	*No*	*100 mg*	*24 hr*
Aminoglycosides			
Gentamycin‡	*Yes*	*(1 mg/kg)*	*48 hr (scr ×8)§*
Kanamycin†	Yes	(7 5 mg/kg)	72 hr (scr ×9)§
Tobramycin‡	Yes	(1 mg/kg)	48 hr (scr ×6)§
Miscellaneous			
Chloramphenicol	No	(12 mg/kg)	6 hr
Clindamycin	*No*	*300 mg*	*12 hr*
Colistimethate	Yes	150 mg	36 hr
Erythromycin	No	400 mg	6 hr

*Can cause acute allergic interstitial nephritis

†Does not cause renal damage but causes elevated BUN by antianabolic effect

‡The aminoglycoside antibiotics are ototoxic as well as nephrotoxic

§The interval between doses in hours can be calculated by multiplying the patient s serum creatinine concentration (SCP) by the factor indicated

because of the accumulation of the drug and danger of toxicity The nephrotoxicity and the need for dose reduction of some commonly used antibiotics are presented in Table 5-2 If it is necessary to use nephrotoxic antibiotics in patients with acute renal failure, serum concentrations of the drug should be monitored to prevent toxicity

The common practice of leaving an indwelling urethral catheter in the bladder of the patient with acute renal failure should be avoided In most cases catheterization of the bladder is not necessary, and only leads to urinary tract infection If there is a valid indication for an indwelling catheter, a triple lumen catheter should be used, and an antibacterial irrigating solution should be constantly infused

CARDIOVASCULAR COMPLICATIONS

Congestive heart failure often develops in patients with acute renal failure Unless the patient has preexisting organic heart disease, this is usually due to salt and water overloading with a resultant overexpansion of extracellular fluid

volume This situation should be managed by dialysis to remove the excess fluid volume Digitalization of these patients is seldom indicated because it often leads to digitalis toxicity, and usually will not relieve the congestive heart failure until the volume overload is corrected

About 25% of patients with acute renal failure will develop hypertension This is usually mild and requires no specific treatment It is due in part to increased extracellular fluid volume and can often be corrected by removing the excess volume by dialysis If antihypertensive medication is required, alpha methyldopa (Aldomet) in a dosage of 750–3000 mg daily is usually effective

Fibrinous pericarditis occurs in about 20% of patients with acute renal failure If a serious effusion forms, or if hemorrhage into the pericardial sac occurs as a result of heparinization during hemodialysis, cardiac tamponade may result The signs of tamponade may be diminution of heart tones, a rising venous pressure, and a falling systemic blood pressure with the appearance of a paradoxical pulse The patient may complain of dyspnea and may demonstrate mental confusion The chest x ray film may show an increase in the size of the cardiac silhouette Once the diagnosis of cardiac tamponade is established emergency pericardiocentesis is indicated If tamponade recurs, pericardiectomy may be necessary

GASTROINTESTINAL COMPLICATIONS

Anorexia, nausea, and vomiting are common symptoms of uremia They are usually corrected by dialysis, and seem to be improved by reducing the protein intake Bleeding may occur from anywhere in the gastrointestinal tract This may be from ulcers or from a diffuse gastroenteritis When ulcers occur, bleeding is aggravated by the coagulation defects associated with uremia Acute pancreatitis may occur, and its diagnosis may be difficult because of the elevated serum amylase levels that are usually present with renal failure

OTHER COMPLICATIONS

Anemia usually occurs in acute renal failure and may appear as early as the first week It is caused by a combination of increased erythrocyte destruction and impaired erythrocyte production The anemia should be monitored closely and transfusions of packed red blood cells administered if the hematocrit falls below 25% Defects in coagulation are commonly encountered in renal failure Most clearly defined is a decrease in platelet adhesion and ADP-induced aggregation Patients with active bleeding may require transfusion with platelet packs or fresh whole blood to establish normal hemostasis

DIALYSIS

The increasing availability of hemodialysis and peritoneal dialysis has facilitated the management of patients with acute renal failure, but the overall mortality for all cases of acute renal failure has not improved significantly since the advent of dialysis However, it has been observed that patients with acute renal failure who were dialyzed before the appearance of uremic symptoms and before the BUN exceeded 100 mg/100 ml had an increased rate of survival

as compared with a group of patients who were not dialyzed until they were symptomatic or until the BUN exceeded 150 mg/100 ml Early and frequent dialysis not only seems to reduce the incidence of uremic complications, but also allows the patient a more generous intake of fluid, calories, and protein, which is beneficial to the patient's recovery, particularly following surgery

The generally accepted indications for either hemodialysis or peritoneal dialysis are as follows 1) symptomatic circulatory overload manifested by congestive heart failure, edema, or serous effusions, 2) hyperkalemia with a serum potassium level over 6 5 mEq/liter that does not respond to the previously mentioned conservative measures, 3) the presence of uremic symptoms, such as anorexia and nausea, neuromuscular irritability, confusion, or a pericardial friction rub, 4) a BUN over 120 mg/100 ml or daily increments of 30 mg/100 ml in patients with severe sepsis or tissue necrosis, 5) severe metabolic acidosis that cannot be controlled with the administration of bicarbonate, 6) the presence of a dialyzable poison or toxic drug

PERITONEAL DIALYSIS VERSUS HEMODIALYSIS

The choice between peritoneal dialysis and hemodialysis is determined by the availability of each of the modalities and the condition of the patient. Peritoneal dialysis has the advantages of greater simplicity, fewer complications, and availability in any hospital regardless of whether a dialysis unit is present Peritoneal dialysis can be performed in the presence of an enlarged, nonpregnant uterus, and in the presence of peritonitis However, late pregnancy, retroperitoneal operations, and the presence of intraabdominal drains contraindicate this procedure

Hemodialysis is more efficient than peritoneal dialysis, and therefore corrects imbalances of fluid and electrolytes more rapidly However, the disadvantages of hemodialysis are that it requires the surgical insertion of an arteriovenous shunt and the administration of heparin to the patient, and it often results in rapid changes in circulating blood volume, which may be poorly tolerated in an unstable patient Although the use of regional heparinization minimizes the risk of bleeding during hemodialysis, peritoneal dialysis is the preferred method of treatment in patients who have a high risk of bleeding, such as patients with peptic ulcer and pericarditis

Following the oliguric phase, which lasts an average of 10–14 days, the patient enters the diuretic phase of acute renal failure The onset of this phase is heralded by a progressively increasing volume of urine Large amounts of water and electrolytes are excreted during this period and the patient's survival demands adequate replacement of these excessive losses Accurate recording of intake and output, daily weights, daily determinations of BUN, creatinine, and of urine and serum electrolytes are important in managing the patient in this phase If the patient can tolerate oral feedings, it is best to replace the fluid and electrolyte losses orally However, if the daily urine output reaches 5–6 liters, it may be necessary to replace these losses intravenously Half-strength Ringer's lactate or 0 45% sodium chloride are the basic solutions Supplemental potassium should usually be added to these solutions to prevent a fall of serum potassium below 3 5 mEq/liter The measurement of the urinary loss of potassium is helpful in determining the amount of potassium supplementation needed

With the onset of the diuretic phase, the azotemia may not improve immediately and the patient may still require dialysis for 4 or 5 more days If the urine volume does not spontaneously decrease after 5–7 days of diuresis, it may be necessary to limit gradually the replacement of fluid loss to determine whether the diuresis is being prolonged by excessive administration of fluids This is best done by reducing the fluid replacement to only 75% of measured losses until the urinary volume decreases to normal The patient s state of hydration must be carefully assessed during this period, because if the patient has lost the capacity to concentrate urine, she may become severely dehydrated when fluid replacement is curtailed

In the recovery phase, the urine volume decreases to normal and the glomerular filtration rate slowly returns toward normal, although many patients never recover completely normal renal function The ability to concentrate the urine returns to normal much more slowly than the glomerular filtration rate, requiring several years in some patients and never returning to normal in others

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Eclamptogenic Toxemia

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Chapter 6

"It is but as a body yet distempered
Which to his former strength may be restored
With good advice and little medicine'
William Shakespeare (1564–1616)
King Henry IV Part Second Act III, Scene I

The term toxemias of pregnancy was formerly applied to a number of conditions manifesting vascular derangements that either antedate or arise during pregnancy or the early puerperium These conditions are characterized by hypertension that may be related to proteinuria, edema, convulsions, coma, and other signs Because the term toxemias has been all-inclusive, discrepancies in statistical reporting have occurred, making it extremely difficult to evaluate the results obtained with different treatment programs In 1972, the Committee on Terminology of The American College of Obstetricians and Gynecologists, after careful consideration and consultation with authorities in the field, suggested classification for this group of conditions hitherto loosely referred to as "toxemias of pregnancy"

1. Gestational edema
   Gestational edema is the occurrence of a general and excessive accumulation of fluid in the tissues of greater than 1+ pitting edema after 12 hours' rest in bed, or of a weight gain of 5 lb or more in 1 week due to the influence of pregnancy
2. Gestational proteinuria
   Gestational proteinuria is the presence of proteinuria, during or under the influence of pregnancy, in the absence of hypertension, edema, renal infection, or known intrinsic renovascular disease
3. Gestational hypertension
   Gestational hypertension is the development of hypertension during pregnancy, or within the first 24 hours post partum, in a previously normotensive woman No other evidence of preeclampsia or hypertensive vascular disease is present The blood pressure returns to normotensive levels within 10 days following parturition Some patients with

gestational hypertension may in fact have preeclampsia or hypertensive vascular disease, but they do not satisfy the criteria for either of these diagnoses

4. Preeclampsia
   Preeclampsia is the development of hypertension with proteinuria, edema, or both, due to pregnancy or the influence of a recent pregnancy It occurs after the 20th week of gestation, but it may develop before this time in the presence of trophoblastic disease Preeclampsia is predominantly a disorder of primigravidas
5. Eclampsia
   Eclampsia is the occurrence of one or more convulsions, not attributable to other cerebral conditions such as epilepsy or cerebral hemorrhage, in a patient with preeclampsia
6. Superimposed preeclampsia or eclampsia
   Superimposed preeclampsia or eclampsia is the development of preeclampsia or eclampsia in a patient with chronic hypertensive vascular or renal disease When the hypertension antedates the pregnancy, as established by previous blood pressure recordings, a rise in the systolic pressure of 30 mm Hg or a rise in the diastolic pressure of 15 mm Hg and the development of proteinuria, edema, or both is required during pregnancy to establish the diagnosis
7. Chronic hypertensive disease
   Chronic hypertensive disease is the presence of persistent hypertension of whatever cause, before pregnancy or before the 20th week of gestation, or persistent hypertension beyond the 42nd postpartum day
8. Unclassified hypertensive disorders
   Unclassified hypertensive disorders are those in which information is insufficient for classification They should compose a minority of the hypertensive disorders in pregnancy

EDEMA

Edema is a general and excessive accumulation of fluids in the tissues, commonly demonstrated by swelling of the extremities and face The fluid may be intracellular or extracellular, and edema is not usually evident until there is a weight gain of 10% Edema is usually physiological but when it occurs with hypertension or proteinuria, perinatal mortality is increased

HYPERTENSION

Hypertension is a rise in the systolic pressure (SBP) of at least 30 mm Hg or a rise in the diastolic pressure (DBP) of at least 15 mm Hg, or the presence of a systolic pressure of at least 140 mm Hg, or a diastolic pressure of at least 90 mm Hg Hypertension may also be signaled by a mean arterial pressure (MAP) of 105 mm Hg or more or by a rise of 20 mm Hg or more MAP = DBP + (SBP − DBP)/3 The levels cited must be manifest on at least two occasions 6 or more hours apart, and should be judged on the basis of previously known blood pressure levels

Data from over 50,000 pregnancies reported by Friedman (1976) have shown that a maternal blood pressure as low as 125/75 mm Hg is ominous if it

represents a significant rise above the prepregnant level Also Page and Christianson (1976) have shown that if the MAP reaches 90 mm Hg, perinatal mortality and postnatal morbidity are increased

PROTEINURIA

Proteinuria is the presence of urinary protein in concentrations greater than 0 3 g/liter in a 24-hour urine collection, or in concentrations greater than 1 g/liter in a random urine collection (1+ or 2+ by standard turbidimetric methods) on two or more occasions at least 6 hours apart The urine must be a clean, voided, midstream specimen, or else obtained by catheterization

Because the other serious hypertensive disorders are amply covered in medical textbooks, the discussion here will be limited to eclamptogenic toxemia This is the condition which alone, or superimposed, presents most problems to the obstetric team

It is generally accepted that "toxemia of pregnancy" is an undesirable term, but there is still no agreement as to what term should replace it In New Zealand the term "HOP syndrome" is used, the letters standing for hypertension edema, and proteinuria When this is translated to the American scene, it would have to be the "HEP syndrome," and this would be unlikely to gain wide scientific acceptance The term "preeclampsia eclampsia" has been suggested, but this is a cacaphonic abomination, and the condition would be best renamed "eclamptogenic disease of pregnancy" However, 'toxemia of pregnancy" has been in widespread use for so long that probably the best we can hope for is adoption of the term "eclamptogenic toxemia"

Eclamptogenic toxemia occurs after the 20th week of pregnancy and is characterized by hypertension, with edema and/or proteinuria, reduced uterine artery flow, reduced renal artery flow, with a reduced glomular filtration rate, possibly some evidence of disseminated intravascular coagulation (DIC), and distinctive renal glomerular changes

In essence, eclamptogenic toxemia refers to preeclampsia and eclampsia

PREECLAMPSIA

Hypertension (BP 140/90 or above, or a rise of 30 mm Hg systolic or 15 mm Hg diastolic, or MAP 105 mm Hg or higher), edema (generalized), and/or proteinuria (300 mg/liter or above in a 24 hour urine specimen)

ECLAMPSIA

Preeclampsia with convulsions and/or coma

INCIDENCE

In the United States (1976) eclamptogenic toxemia complicates 1.5% of pregnancies among patients delivered in private hospitals It complicates 10–15% of pregnancies in patients delivered in public hospitals or university teaching hospitals The overall occurrence rate in the United States is approximately 5% of all pregnancies

IMPORTANCE

Eclamptogenic toxemia is important for at least four reasons

1. It is the second leading cause of maternal mortality in the United States
2. It is a major cause of perinatal mortality, being associated with approximately 30,000 perinatal deaths per year in the United States
3. It is associated with an increased tendency to intrauterine growth retardation in the fetus
4. It is associated with an increased tendency to mental retardation in surviving offspring

ETIOLOGY

The cause of eclamptogenic toxemia remains unknown despite extensive investigations The causative factors most commonly identified are listed according to the evidence at hand

1. Dietary Factors The geographic distribution of eclampsia suggests that diet plays an important part in the etiology Multiple dietary factors have been incriminated including hypoproteinemia thiamine deficiency, calcium deficiency, iron and vitamin deficiency, and excess of carbohydrate and sodium in the diet
2. Endocrine Dysfunction During normal pregnancy, enlargement of the anterior pituitary, adrenal, thyroid, and parathyroid glands occurs Interference with hormonal activity or metabolism by the developing placenta has also been postulated.
3. Allergic Phenomena Interest in this aspect had been waning until recently, when immunofluorescent studies identified immunoglobulins in the tissues of women with toxemia of pregnancy
4. Toxic Manifestations Water intoxication, menotoxin, and miscellaneous other agents have been blamed
5. Hemodynamics Hypothesis Eclamptogenic toxemia and placenta previa rarely coexist It has been suggested that when the placenta is implanted in the upper uterine segment, then much of the venous blood returns to the heart via the ovarian veins In traversing this route, the additional blood volume encourages congestion of the renal, hepatic, and cerebral venous systems and results in hypoxic changes Another hemodynamics theory is that the cause of eclamptogenic toxemia is hypovolemia, which leads to a hypoperfusion syndrome The weight of evidence suggests, however, that hypovolemia, like DIC, is an important part in pathogenesis but is not the primary cause of eclamptogenic toxemia
6. Stress Reaction This, too, is not so popular a theory as it once was
7. Uterine Stretch Reflex It has been suggested that the resistance of the myometrium to stretching initiates a uterorenal reflex resulting in renal cortical ischemia This in turn, has been said to result in generalized vasoconstriction, with hypertension, proteinuria, and edema While this is clinically appealing because of the association between eclamptogenic toxemia and such factors as multiple pregnancy, the weight of experimental evidence on pathogenesis is against this theory

Factors that are commonly associated with eclamptogenic toxemia are primi-

gravidity, multiple pregnancy, polyhydramnios, vascular disease, trophoblastic disease, and abruptio placentae

Eclamptogenic toxemia has a geographic distribution that is not entirely related to the quality of obstetric care, and the condition is sometimes seen in the immediate postpartum period

PATHOGENESIS

Spiegelberg, in his textbook of obstetrics published in 1878, was probably the first obstetrician to mention a relationship between the uterus and toxemia of pregnancy He suggested that irritation of the uterine nerves may possibly lead to renal arteriolar vasospasm In puerperal eclampsia immediate removal of placental tissue and coagulated blood which might act as a reflex stimulus, was recommended Thus, Spiegelberg deserves the credit for originating the utero-renal reflex theory later fostered by Sophian (1949, 1953, 1955, 1957, 1958, 1959, 1961)

The placental concept was proposed by Young in 1914 and pursued by the same investigator in 1927 He was impressed by the frequency of infarcts in toxemic placentas and postulated that this process was associated with utero-placental ischemia Young felt that toxemia was due to autolytic products formed in necrotizing areas in the placenta, on the basis of experiments in which guinea pigs were given injections of material squeezed from autolyzed placentas In deed, here was the germ of the thromboplastin concept Bartholomew *et al* (1957) also stressed the importance of placental necrosis in forming degradation products with toxic effects for the mother They were of the opinion that oxytocin acted as a spasmogenic agent on the placental vein sphincters and initiated infarction of the placenta by this mechanism. The idea of placental autolysis as an important etiologic factor has also been supported by Magara (1960), who suggested that hypercholesteremia encouraged placental degeneration and that this was followed by autolysis and the liberation of a toxic vasoconstrictor polypeptide

In 1929 Beker, in Holland, being impressed with the increased frequency of toxemia in the primigravida, injected barium into the uterine arteries of primigravid and multiparous cows during pregnancy In the primigravid cows the vessels were much narrower In 1948, he demonstrated similar results using human uterine specimens and concluded that in toxemia the hypertension developed in an attempt to maintain an adequate uteroplacental blood flow

The etiology of pregnancy toxemia was further elucidated by Ogden, Hildebrand, and Page (1940) These workers were able to produce hypertension in pregnant dogs by partially clamping the abdominal aorta below the level of the renal arteries However, no rise in blood pressure was observed when the same procedure was carried out after removal of the uterus and its contents Control experiments in nonpregnant dogs revealed that clamping of the aorta caused no hypertension Thus, the conclusion was reached that changes in the fetus, the placenta, or the gravid uterus were responsible for the increase in blood pressure of the pregnant animals

Van Bouwdijk Bastiannse *et al* (1954), in similar experiments, on rabbits which had their ovarian arteries ligated previously, noted a rise in blood pressure in pregnant animals after partial clamping of the uterine arteries In nonpreg-

nant animals, the uterine ischemia obtained did not lead to hypertension The work of these investigators suggested that placental rather than uterine ischemia was the cause of the hypertension and that it represented an effort by the mother (or perhaps the fetus) to improve uteroplacental blood flow

Of great interest in this respect is the reported coexistence of toxemia in women with aortic hypoplasia and with hypoplasia of the iliac vessels

Another interesting contribution was made by Thompson and Tickner (1949), who reported that the placenta was rich in monoamine oxidase but that placental hypoxia resulted in its inactivation This enzyme is highly sensitive to oxygen tension and has the ability to destroy vasoconstrictor amines such as norepinephrine epinephrine and tyramine In 1958 Browne postulated that pregnancy produced a Cushingoid syndrome and that deficiency of monoamine oxidase in an ischemic placenta could result in hypertension because of the unopposed action of endogenous vasoconstrictor amines

In 1952 Assali *et al* established beyond reasonable doubt that the hyper tension in toxemia was of humoral not neurogenic origin

The proof of the existence of placental ischemia in clinical toxemia of preg nancy was given by Browne and Veall (1953) who demonstrated by the use of radioactive sodium (^{24}Na) a reduction in the placental blood flow to approxi mately one third of normal in patients with preeclampsia However since similar results were obtained in chronic hypertension they felt that the placental ischemia was the result rather than the cause of the hypertension In their dis cussion they cited an exacerbation of a preexisting hypertension by placental ischemia as an additional mechanism which would account for the known tendency of the chronic hypertensive to develop a superimposed preeclampsia

Morris *et al* (1956) also using radioactive sodium found that uterine blood flow was reduced in proportion to the severity of preeclampsia

Hunter and Howard (1961) reported on the existence of a heat stable vaso-pressor agent (hysterotonin) in the decidua and amniotic fluid of toxemic patients They believed the decidua to be the site of production of this hyper tensive substance but although it was also found in the maternal plasma it could not be found in the placentas of toxemic women

On the other hand Neuweiler *et al* (1958) were successful in isolating a toxemia producing fraction from toxemic placentas Their work has been con firmed and further pursued by Rubinstein (1962) This same pressor com ponent was found by Berger *et al* to be in the polypeptide fraction although with the same method the fraction could not be found in the placentas of healthy women However the existence of pressor substances in toxemic placentas is not absolute proof of their placental origin

Gyongyossy and Kelentey (1958) induced hypertension in pregnant cats by distending the uterine horns with balloons introduced into the amniotic sacs In addition they were able to transfer a vasopressor substance from these animals to other cats which in turn became hypertensive Since these experiments were not performed in nephrectomized animals however the conclusion that the pressor agents were of placental origin is not justified

The fact that toxemia is more likely to occur in the primigravida and in women with multiple pregnancy or polyhydramnios has led to the general ac ceptance of a cause-effect relationship between increased intrauterine pressure and toxemia of pregnancy Sophian s uterorenal concept of the etiology of toxemia has been a thought provoking contribution He postulated that it is not

merely the stretching, but the inherent resistance of the myometrium to stretching that leads, by way of a neurogenic reflex, to renal cortical ischemia Sophian has maintained that, as a result of this renal ischemia, pressor substances are formed in the kidneys and it is these substances which are responsible for the development and persistence of hypertension

In 1962 Kumar developed hypertension in dogs in a chronic experiment involving uteroplacental ischemia In 1963 Berger and Cavanagh developed experimental hypertension and proteinuria in pregnant rabbits by suture infarction of the placentas From this series of experiments three main conclusions were drawn 1) Placental ischemia but not uterine ischemia is essential for the production of experimental hypertension in pregnant rabbits at term 2) In transient placental ischemia produced by compression of one gravid horn of the rabbit uterus, there is a neurogenic reflex that causes an evanescent rise in blood pressure In prolonged placental ischemia obtained by placing Z sutures through the placentas, a humoral factor is responsible for the persistent increase in blood pressure 3) The kidneys are not necessary for the production of experimental hypertension in rabbits Placental ischemia leads to experimental hypertension even in the absence of the kidneys, so it is evident that the vasopressor substances are not of renal origin Although it was felt that the vasopressor agent was probably of placental origin, this could not be stated unequivocally

In 1967 Hodgkinson, Hodari, and Bumpers devised a very satisfactory chronic toxemia model in the dog by placing Teflon bands around the uterine arteries and transecting the ovarian arteries in the nonpregnant state Those animals that became pregnant following this procedure developed hypertension and proteinuria

In 1859 James Young Simpson wrote "Albuminuria, dropsy, and convulsions are successive effects of one common cause, *viz.* a pathologic state of the blood, to the occurrence of which pregnancy in some way peculiarly predisposes" In 1948 Page postulated that the placental dysfunction in eclamptogenic disease led to the liberation of thromboplastin, which became one of the mediators of the disease However, McKay *et al* were the ones who established this concept over the period 1953–1972 In 1953 they drew attention to the similarity between eclampsia and the generalized Shwartzman reaction In 1967 they reported on the production of "experimental eclampsia" in rats by means of a lipid peroxide diet, concluding that the source of thromboplastin initiating the DIC was the blood platelet

Disseminated intravascular coagulation or more accurately intravascular coagulation-fibrinolysis syndrome (ICF), has been described in association with eclamptogenic toxemia Pritchard *et al* (1976), and Bonnar *et al* (1971) studied patients with severe preeclampsia The most consistent findings were of thrombocytopenia and an elevation of serum fibrin degradation products Beacham, Watson, and Clapp (1974) found fibrinogen levels only slightly depressed or normal in the face of severe fibrin deposition Roberts and May (1976) found evidence of consumptive coagulopathy in 9 1% of eclamptic patients and in 2 6% of patients with severe preeclampsia Pritchard *et al* (1976) found thrombocytopenia in 29%, a prolonged thrombin time in 50%, abnormally elevated serum fibrin degradation products in 3%, and circulating fibrin monomer in 5% of eclamptic patients studied In view of the degree of change in the maternal clotting mechanism as compared to that seen in severe

abruptio placentae and prolonged retention of a dead fetus, these workers concluded that the coagulation changes, when present in eclampsia are effect rather than cause Also, if heparin is to be used in the management of eclamptogenic toxemia, it should be used only with strict laboratory control, otherwise, more harm than good will come of it

In trying to understand the pathogenesis of eclamptogenic toxemia, one other factor must be appreciated Whereas in normal pregnancy there is a reduced vascular response to vasopressor substances in eclamptogenic disease the response is increased As long ago as 1937, Dieckmann and Michel reported a marked increase in the vascular response of toxemic women to vasopressin Talledo *et al* (1968) have shown a similar response to angiotensin and norepinephrine The actual concentration of angiotensin in the plasma is about the same in normal pregnancy as in toxemia, and the difference in response is probably brought about by increased concentrations of sodium in the mucopolysaccharides of the arterial wall The work of Burks *et al*, (1971) who studied isolated arteries appears to confirm this view It is clear, therefore that generalized vasoconstriction probably plays a very important part in the pathogenesis of eclamptogenic toxemia and that this is associated intimately with the renin angiotensin aldosterone system

It must be appreciated that normal pregnancy is a high renin state Plasma renin activity plasma renin concentration renin substrate (angiotensinogen), and plasma angiotensin II are all above normal Renin released into the blood stream has a half life of about 2 hours in nephrectomized subjects (Browne *et al* 1969) It is a proteolytic enzyme devoid of vasoactive properties It acts upon circulating α_2 globulin angiotensinogen to yield the decapeptide fraction angiotensin I Angiotensin I is then converted to angiotensin II by the action of the converting enzyme in the lung which splits off the two terminal amino acids of angiotensin I Angiotensin II is the most potent vasopressor known Its production occurs predominantly in the pulmonary circulation but it also occurs in the kidney and to a lesser extent in the circulating plasma Angiotensin II is degraded by a group of enzymes collectively called angiotensinase, hence the half life of angiotensin II in the plasma is less than 3 min (Vane 1969) In preeclamptic toxemia the plasma renin concentration and plasma renin activity tend to be decreased although most of the values fall within the range seen in normal pregnancy Plasma renin sensitivity is either unchanged or slightly decreased relative to normal pregnancy Plasma levels of angiotensin II are low as compared even to nonpregnant women and are severely depressed in comparison with normal pregnancy

Normal pregnant women are highly resistant to the pressor and renal effects of infused angiotensin II Women with eclamptogenic toxemia are very sensitive to its pressor effect but markedly resistant to its renal effect (Chesley 1975) Women who will develop preeclampsia can be identified by their progressive loss of resistance to exogenous angiotensin II This occurs many weeks before any recognizable sign of toxemia appears and forms the basis of a clinical test for high risk pregnancy (Gant *et al* 1973)

The part played by prostaglandins is unclear at this time but it has been suggested that they may be important (Speroff 1972) Prostaglandin E (PGE) may be involved because it has been found that during the third trimester it is released into the uterine veins during infusion of angiotensin II in the rhesus

monkey (Franklin *et al* 1974). Terragno *et al* (1974) infused angiotensin II into pregnant dogs and found that the PGE level in uterine venous blood almost doubled, and the uterine blood flow was increased. When the prostaglandin inactivator indomethacin was given, the uterine blood flow fell to about 25% of the control level, and PGE almost disappeared from the uterine venous blood. These data suggest that PGE may regulate uteroplacental blood flow.

Demers and Gabe (1976) have reported that placentas from toxemic women showed decreased PGE (vasodilator) and increased prostaglandin F (vasoconstrictor). Their data suggest that prostaglandin synthesis and/or metabolism is altered in preeclampsia. Whether this abnormality is a primary event or is secondary to other regulatory mechanisms is not clear at this time. In the nonpregnant human uterus, the blood flow to the endometrium is in the range of a few milliliters per minute, whereas in the third trimester of pregnancy approximately 500 ml/min are required for the fetus and placenta. To achieve this marked increase in blood flow, hypertrophy of the uterine vasculature occurs (Brosens *et al* 1967). Acute atherosis has been reported in the myometrial segments of the uteroplacental arteries in toxemic patients (DeWolf, *et al* 1975). In the patient who develops toxemia in the third trimester, the toxemic vascular lesion—comprising decidual arteriolar thickening, narrowing, trophoblastic degeneration, and fibrin thrombi deposition on chorionic villi—may be present as early as the first trimester of pregnancy (Nadji and Sommers, 1973). Thus, the partial occlusion of the uterine arteries with transection of the ovarian arteries either before or during pregnancy is a logical step in the production of uteroplacental ischemia. This is, of course, an extension of the concept of Goldblatt *et al* (1939) and assumes that, if chronic renal ischemia leads to hypertension, then chronic uteroplacental ischemia may lead to hypertension. In 1976, Abitbol *et al* succeeded in producing a toxemia model by constricting the aorta below the level of the renal arteries in rabbits. They reported the occurrence of hypertension, proteinuria, and increased weight gain. Also, they demonstrated evidence of a lesion similar to that found in clinical toxemia in the kidneys by light microscopy, electron microscopy, and immunofluorescence. Examination of the placentas of these animals revealed diffuse congestion, infarction, and syncytial knots resembling the histopathologic picture in human toxemia. Using the same technique of partial aortic occlusion with a number 3 silk ligature, hypertension and proteinuria were produced in pregnant dogs, findings on light microscopy, electron microscopy, and immunofluorescence revealed renal lesions similar to those found in human toxemia (1976). The most important animal model that could be developed would be in the subhuman primate, but this has presented a great many difficulties.

In 1970, Misenhimer *et al* reported on an experimental model of chronically impaired uterine artery flow in the rhesus monkey. These workers did not measure blood pressure, but angiographic studies showed a marked reduction in intervillous space inflow in some animals, the fetal death rate was 60%, with all deaths occurring near the beginning of the third trimester of pregnancy.

If the uteroplacental blood flow is decreased, the uterine renin-angiotensin system will be activated, and there is some evidence that plasma renin levels are higher in the uterine vein than in the peripheral circulation in human eclamptogenic toxemia. Thus, in toxemia, release of angiotensin II from the uterus may be stimulated by a decrease in uteroplacental blood flow, and this circulating

angiotensin II may suppress renal renin release This could explain the apparent paradox of renin angiotensin levels in toxemia However, clarification of this hypothesis would entail simultaneous measurement of renin angiotensin levels in renal and uterine venous blood This is not feasible in human pregnancy but can be studied in the subhuman primate if a suitable model can be developed

Over the period 1968–1976 we have conducted a multidisciplinary program in our laboratory with a view to developing a toxemia animal model in a subhuman primate We have published reports on these studies elsewhere (1972, 1974, 1977), so here we will merely summarize our findings Baboons were used in this study, because their reproductive physiology is remarkably similar to that of human subject, and this certainly would make the findings more meaningful

In our baboon model we have observed the development of hypertension, proteinuria, and a reduction in renal artery flow in pregnant study animals whose ovarian arteries had been transected, and whose uterine arteries had been partially occluded by metal clips *prior to* conception In addition fibrin/fibrinogen deposits were confirmed in the pregnant study group, using immunofluorescence studies, although no evidence of DIC could be found in four animals on which extensive coagulation profiles were made Furthermore, we have found that when, using the same experimental technique, uterine ischemia is produced in baboons *during* pregnancy, these animals also develop hypertension and proteinuria Studies by light microscopy, electron microscopy, and immunofluorescence revealed that the renal lesion in the toxemic baboon is indistinguishable from that in women with eclamptogenic toxemia After 8 years of work on this animal model, we feel justified in concluding that although refinements are required, a primate experimental model is now available for studying the pathogenesis of, and for evaluating new methods of treatment for, eclamptogenic toxemia

In the present light of our knowledge, it appears that uteroplacental ischemia is the "trigger mechanism" for initiating toxemia, not only in the rabbit and dog but also in the primate The more important points in pathogenesis are presented in Figure 6-1

PATHOLOGY

In 1954, Donnelly and Lock investigated the cause of death in 533 women with toxemia of pregnancy They found that death was attributable directly to toxemia in 393 cases The most common causes of death were cerebral vascular accidents, cardiopulmonary failure, and acute renal failure

The lesions usually found in eclamptic patients who die, and in preeclamptic or eclamptic patients in whom renal biopsies are performed are as follows

Kidney

The characteristic lesion in eclamptogenic toxemia is a renal glomerular endotheliosis The endothelial cells are swollen and the amorphous material deposited in the cytoplasm causes enlargement of the capillary tufts The lumens of the glomerular capillaries become narrow, so that ischemia is the result of organic

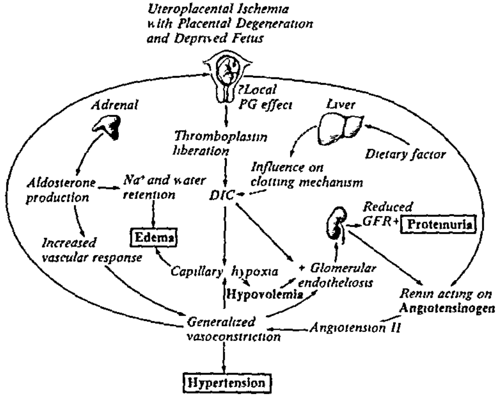

FIG 6 1 Pathogenesis of eclamptogenic toxemia

narrowing as well as vasospasm These changes are the ones that markedly reduce the glomerular blood flow and the glomerular filtration rate

Immunofluorescent techniques have demonstrated the presence of fibrin/fibrinogen deposits, immunoglobulins and complement in the glomerular mesangium and capillary vessel walls Renal tubules usually show abnormalities consistent with ischemia, and proteinaceous material is often noted within the tubular lumens

In the patients who survive, complete repair of the glomerular lesion following delivery is the rule Nevertheless some patients have been reported as showing evidence of glomerular damage months or even years after delivery

In very rare instances severe renal vascular spasm may produce extensive arterial thrombosis resulting in bilateral renal cortical necrosis This is especially likely to occur in patients with eclamptogenic toxemia complicated by abruptio placentae

Liver

Significant liver damage may occur in eclamptogenic toxemia In eclampsia about 75% of the patients show some evidence of hepatic dysfunction, but permanent damage is very rare The most common hepatic lesion in the eclamptic patient is periportal hemorrhagic necrosis, this may extend toward

the center of the hepatic lobule The surrounding blood sinuses may be c pressed In some areas, extravasation may occur and fibrin clots may fo especially at the bases of the liver cell columns These changes are believed t the result of thrombosis in the hepatic arterioles

Hemorrhage under the capsule of the liver may occur in severe preeclam and eclampsia, and even consequent intraabdominal bleeding has been repo as an acute surgical emergency

In 1976, Arias and Mancilla Jimenez reported immunofluorescent evid of fibrin/fibrinogen, immunoglobulins, and complement in the livers of pati with eclamptogenic toxemia Fibrin/fibrinogen outlining the hepatic sinus was found in 12 patients with preeclampsia, and in two there were large nod deposits of fibrin/fibrinogen deposits of immunoglobulins G and M (IgG, Ig and of the heat-stable component of complement (C'_3) in areas of necr These investigators postulated that changes in the liver and kidney were evi in patients with eclamptogenic toxemia because the generalized vasosp characteristic of toxemia is more severe in these organs They pointed out the increased vasospasm, in the presence of a systemic yet mild blood hy coagulability, would create adequate local conditions for the precipitatio fibrin/fibrinogen

Brain

Edema and hemorrhage are commonly seen in patients dying of eclampsia patients who survive, cerebral edema is common, and it is estimated that s cerebral hemorrhages may occur in almost 20% of women with eclamptog toxemia The pons, basal ganglia, and subcortical areas are most often invol If the patient recovers from eclampsia, residual brain damage is rare, althou few cases of paralysis and pituitary necrosis have been reported

Lungs

In patients who have died of eclampsia, there is almost always evidence pulmonary edema and diffuse hemorrhagic bronchopneumonia

Heart

In the myocardial tissue of women who have died of eclampsia subendothe hemorrhages, fibrin thrombi, and focal necrosis may be found and are someti so severe as to cause discoloration in the heart This damage to the heart h to explain related pathophysiologic changes, such as impaired cardiac rese and arrhythmias

Placenta

Histopathologic changes in the placenta of patients with toxemia reveal p mature aging of the placenta, syncytial degeneration and congestion of intervillous spaces Degeneration and thrombosis of the spiral arterioles in decidua indicate an acute atheroma Red infarction is the typical gross lesio the placenta of a patient with eclamptogenic toxemia, and is present in ab 60% of the patients

PREVENTION AND DIAGNOSIS

Although eclamptogenic toxemia often cannot be prevented, it is possible to select groups of patients who are particularly prone to develop the disease and to monitor these patients carefully for predisposing factors and early signs of developing toxemia

- Nulliparity
- Multiple pregnancy
- Hydatidiform mole
- Chronic hypertension
- Chronic renal disease
- Diabetes mellitus
- Hydrops fetalis
- Malnutrition

The first three factors are beyond the control of the obstetrician, but meticulous control of diabetes during pregnancy, with frequent hospitalization, if necessary, may help to decrease the adverse effects of this disease on mother and fetus Hydrops fetalis is largely preventable today, and careful monitoring and treatment of the patient with Rh disease will help reduce the incidence of toxemia in chronic hypertension and renal disease

NUTRITION

The role of malnutrition in the genesis of toxemia has already been discussed in the section on Etiology earlier in this chapter T H Brewer has written extensively on the importance of a good diet in reducing the tendency to hypovolemia and a hypoperfusion state In a group of 7000 underprivileged patients in whom particular attention was paid to nutrition, he reported a toxemia rate of 0 55% with no cases of eclampsia This is a favorable result in comparison to the 10–15% incidence usually seen in such patients

BLOOD PRESSURE MEASUREMENT

The blood pressure should be routinely checked at each prenatal visit, and of course a significant rise indicates developing toxemia A mean arterial pressure in excess of 92 mm Hg during the second trimester is a good indicator that the patient will subsequently develop toxemia However, in many patients who develop toxemia, the mean arterial pressure does not attain this level in the second trimester, and toxemia may develop rapidly, without symptoms The need for a test to detect those patients who will develop the disease is obvious The roll-over test may meet this need

Roll-over Test

This simple office procedure was described by Gant *et al* in 1974 The patient is initially placed in the lateral recumbent position and her blood pressure checked until it has stabilized The patient is then turned into the supine position and the pressure again checked. An increase of 20 mm Hg or more in diastolic

pressure is a positive response The test is not reliable after 32 weeks' gestation

Gant *et al* found that patients in whom the roll over test was positive were very sensitive to angiotensin infusion They also found that 98% of these patients subsequently developed toxemia, whereas 91% of the patients in whom the test gave negative results did not become hypertensive during the pregnancy

Gusdon *et al* (1977), however, were more conservative in their assessment of the test. They found that negative test results were an accurate (93%) indicator that toxemia would not develop, but that positive results accurately predicted the subsequent development of toxemia in only 50% of primigravidas and 25% of multiparas

Further study is required before the true value of this promising test can be assessed

EDEMA

Traditionally, edema has been described as the earliest sign of developing toxemia However, edema, by itself, like increased plasma volume, is to be expected during pregnancy It is physiologic and may even be protective against the development of toxemia Eighty-five percent of patients who develop generalized edema have perfectly normal pregnancies only 15% develop toxemia Thus, the concept that treating edema will prevent toxemia is invalid In fact, some methods particularly the administration of diuretics, may even be harmful The administration of diuretics excessively reduces the metabolic clearance rate (MCR) of dehydroisoandrosterone sulfate (DS) in relation to maternal weight loss As diuretics decrease plasma volume, a decrease in the MCR_{DS} probably reflects reduced placental perfusion (Gant *et al* 1975) Both of these factors—decreased plasma volume and reduced uteroplacental perfusion—are thought to be important in the pathogenesis of toxemia, so diuretic administration may actually predispose the patient to the development of the disease These considerations virtually contraindicate the use of diuretics in pregnancy unless there are compelling reasons

PROTEINURIA

Proteinuria is usually the last of the clinical triad to appear It is also the most ominous A combination of 2+ proteinuria (1 g/liter) and hypertension at least doubles the perinatal mortality (deAlvarez, 1976) Edema with proteinuria also increases perinatal mortality (Vosburgh, 1976) Even a level of 1+ proteinuria (300 mg/liter) in the absence of contamination of the specimen or urinary tract infection should be considered significant, particularly when the diastolic blood pressure is 85 mm Hg or higher Because proteinuria may be variable, it is advisable to measure it in a 24-hour urine collection

MANAGEMENT

The obstetrician must always remember that he is treating two patients, mother and fetus, and it is often difficult to ensure that the best interests of both are served The definitive treatment of toxemia is delivery of the infant, all other

forms of treatment are merely supportive The objective of supportive management is to ensure both maternal and fetal well being

CARE OF THE MOTHER

Two facts should be borne in mind when managing patients with toxemia 1) Maternal mortality in preeclampsia is minimal, but maternal mortality from eclampsia is appreciable and increases with the number of seizures, 2) the most common single cause of death in eclampsia is massive cerebral hemorrhage

Prevent Convulsions

BED REST Bed rest by itself may have an antihypertensive effect, and bed rest in the lateral recumbent position is said to increase uterine blood flow, an effect especially beneficial in eclamptogenic toxemia As has been shown by Lauth *et al* (1976), excellent results can be achieved in primigravidas with pregnancy-induced hypertension, "with good advice and little medicine ' The 346 patients were all significantly hypertensive, but only 50% had edema, and only 13% had proteinuria Nevertheless, these patients monitored prior to term in a high-risk pregnancy unit were allowed a normal diet and ambulation *ad libitum* They received no sedatives, antihypertensive drugs or diuretics Their blood pressure was checked four times daily, their urinary protein and weight three times weekly, creatinine clearance weekly, with serial sonography to monitor fetal growth Amniocentesis was used only four times for lecithin/sphingomyelin ratio, and the oxytocin challenge test was not used (this test is defined later in this chapter, in the section on Fetal Reserve) Delivery was delayed until term unless deterioration was evident With this method of management, the perinatal mortality was 9/1000 Among the 26 women who left the unit against medical advice, the perinatal mortality was 154/1000 In view of this report and the report of Fleigner (1976), we should probably reassess the view, so long held, that prompt delivery is the best solution for women with hypertensive disorders of pregnancy With conservative care the prematurity rate will be reduced and if the patients are carefully monitored, there is apparently no hazard to mother or baby

If, in spite of the simple course of management described, the disease worsens, or if the patient is first seen when suffering from severe toxemia more aggressive management is indicated All persistently symptomatic patients should be placed in this group (Table 6-1) In addition, any patient with a diastolic blood pressure of 110 mm Hg (measured on two occasions 6 hours apart with the patient in bed), or proteinuria 3+, or oliguria (400 ml urine or less in 24 hours), or pulmonary edema, or cyanosis would be classified as severe. Patients with severe disease should be nursed in a quiet, darkened room

SEDATIVES AND ANTICONVULSANTS The object of treatment with these substances is to prevent the convulsions of eclampsia

Magnesium Sulfate The magnesium ion has for many years been the cornerstone of treatment of severe toxemia Its main effect is at the neuromuscular junction, but it also decreases cerebral edema It must be administered parenterally, but

TABLE 6-1 Symptoms of Impending Eclampsia

Symptom	Cause
Severe headache	Cerebral edema
Visual disturbance (scotomas diplopia blurred vision)	Cerebral edema
Vomiting	Cerebral edema
Epigastric pain	Stretching of liver capsule or subcapsular hemorrhage

TABLE 6-2 Dosage Schedules for Magnesium Sulfate

	Intravenous	Intramuscular
Initial dose	2-4 g $MgSO_4$ (USP) as 10% solution injected slowly over 2- to 4 min period	10 g $MgSO_4$ (USP) as 50% solution in H_2O divided doses 2 ml in each buttock 1% procaine may be added to reduce pain of injection
Maintenance dose	20 g $MgSO_4$ in 1000 ml 5% dextrose in H_2O usual maintenance dosage 1 g/hr depending on reflexes respirations urinary output etc	5 g $MgSO_4$ as 50% solution every 4 hr (administered same as initial dose) depending on reflexes etc

may be given either intramuscularly or intravenously The advantages of intra venous administration are as follows 1) Rapid onset of effect, 2) optimum dosage may be easily determined for individual patients, 3) may be rapidly stopped in the event of overdosage, 4) nonpainful injection Dosage schedules are presented in Table 6-2

The patient must be vigilantly observed for evidence of overdose, which will manifest itself initially as sluggish knee and biceps jerks and later as depressed respirations and decreased urinary output If not treated, the patient may go into respiratory failure, or at very toxic levels, cardiac arrest may occur Slowing or stopping the infusion will suffice for mild evidence of overdose, but evidence of severe overdose (respirations below 14/min) should be treated by immediate intravenous injection of 10 ml of 10% calcium gluconate solution as a bolus Such a solution should always be kept beside the bed when magnesium sulfate therapy is being used

Barbiturates Barbiutrates have both sedative and anticonvulsant effects, and may be used as an alternative to magnesium sulfate They are particularly useful in the patient who has had one or more convulsions The usual dosage is amo barbital (Sodium Amytal), 0 25–0 5 g, as a slow intravenous injection This may be repeated as necessary to prevent the patient's becoming too restless

Diazepam Diazepam (Valium) has an anticonvulsant effect when given intravenously, but it often has to be given in very large maintenance doses It crosses the placenta, and the neonate cannot excrete the drug, toxic levels have been detected in the infant as long as 3 weeks after delivery It is not, therefore, recommended except occasionally for postpartum eclampsia

Morphine Magnesium sulfate and barbiturates have no analgesic effect, so morphine may be considered for both its sedative and analgesic effects, especially in early labor and after delivery The usual dose is morphine sulfate, 7 5 mg intravenously, repeated as necessary Some fetal depression is inevitable but a pharmacologic antagonist is available (see chapter 13)

Prevent Cerebral Hemorrhage

The drugs that have been mentioned have a mild antihypertensive effect, but this is insufficient for patients with severe hypertension

Hydralazine This drug (Apresoline) acts by causing arteriolar vasodilation It also increases cardiac output and renal blood flow and is thought to increase placental blood flow Hydralazine, 20–40 mg in 1000 ml of 5% dextrose in 0 45% saline, should be infused slowly if the blood pressure is in excess of 160/100 mm Hg The rate of flow of the intravenous fluid should be titrated to the patient's blood pressure The latter should be stabilized at a level consistent with an adequate urinary output The fetal heart should be carefully monitored during administration, for a sudden fall in blood pressure may cause severe fetal hypoxia The most common side effect seen is tachycardia, which may be severe and may occasionally progress to arrhythmias This effect may be abolished by administration of propranolol intravenously

Diazoxide This drug (Hyperstat) has been found to be remarkably successful in treating acute hypertensive states It may, however, cause sudden hypotension which can cause severe fetal hypoxia, and the dose cannot be titrated because the drug is only effective when given as a bolus injection This objection does not apply to severe postpartum toxemia, for which it is a very useful drug The usual dose is 300 mg as a rapid intravenous injection

Expand Plasma Volume

The plasma volume in toxemia is depleted and recently it has been suggested that severe preeclampsia and eclampsia should be treated as a hypoperfusion syndrome, with chlorpromazine to combat vasoconstriction and dilate the vascular bed, while adequate volume replacement with albumin and plasma expanders is carried out (Soder, *et al* 1975) This regimen together with oxygenation, and measures to combat acidosis, has much to recommend it, but unless careful central venous pressure or pulmonary artery wedge pressure monitoring is carried out, pulmonary edema may be precipitated in some patients

Treat Complications

The most serious complications of toxemia are abruptio placentae, pulmonary edema acute renal failure, eclampsia cerebral accident, and cardiac failure Once one of these is present the patient should be delivered as soon as her condition has been stabilized

Abruptio placentae is discussed in Chapter 8, acute renal failure is the subject of Chapter 5, and eclampsia is discussed in detail later in the present chapter

PULMONARY EDEMA Acute pulmonary edema is a very serious medical emergency, and patients in whom it develops should be admitted to an intensive care unit The patient should be given oxygen, morphine, and digoxin in addition to magnesium sulfate and hydralazine, if necessary Furosemide (Lasix) is a very useful drug in treating pulmonary edema but theoretic objections have been raised to its use in toxemia induced pulmonary edema because of the concomitant low plasma volume, and low plasma colloid osmotic pressure On the other hand, increased peripheral resistance and systemic hypertension may lead to left ventricular failure and pulmonary edema Under ideal circumstances therefore the use of furosemide and the infusion of colloid or crystalloid solutions should be governed by the pulmonary artery wedge pressure (see Ch. 4 Life Threatening Infections)

CARE OF THE FETUS

In cases of eclampsia and in cases of severe toxemia which do not respond to conservative therapy as outlined above the infant should be delivered, but in mild to moderate toxemia delivery may often be deferred until conditions are more favorable Since there is no danger to the mother in these cases provided her condition does not deteriorate, a decision to intervene will be made only in the fetal interest Clinical methods of assessing the well being of the fetus are not very accurate and so a number of laboratory tests have become necessary Only those in general use will be discussed

Fetal Growth

Adequate fetal growth is a good indicator of fetal well being Growth may be estimated by palpation and weekly measurements of fundal height but such estimates are often inaccurate Ultrasonic cephalometry has been shown to be an accurate method of assessing fetal growth but it must be done serially starting at 20 weeks gestation Cessation of growth is a good indicator of poor placental function

Fetoplacental Unit

Measurements of estriols in maternal urine or plasma indicate the status of both fetal and placental function and may be affected by a variety of factors (Fig 6 2) Nonetheless they remain the most reliable tests of the fetoplacental unit Twenty four hour urinary estriol estimations have been used successfully all over the world for this purpose (MacLeod *et al* 1971 Fleigner 1976 Garoff and Seppala 1976) It has been suggested that a morning urine estriol estimation is more convenient and may be as accurate as the 24 hour specimen (Bierenski Millner, 1976) Plasma estriol (Klopper, Masson 1975) has also been used with success and is to be preferred in patients with underlying renal disease Falling levels of estriols are a good indicator that the fetus is in jeopardy

Placental Function

The debate whether human placental lactogen reflects placental function or is merely an index of placental size and weight has not been settled Nonetheless it has been successful in predicting failing placental function (Spellacy *et al* 1975 Trolle *et al* 1976)

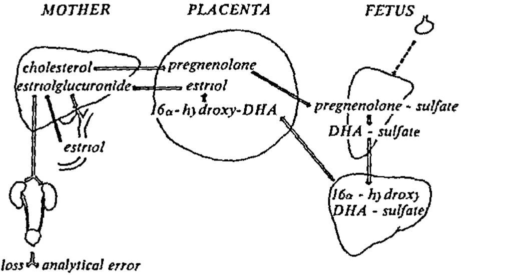

FIG 6-2 Estriol biosynthesis in the last part of pregnancy (Trolle D Bock JE Gaede P The prognostic and diagnostic value of total estriol in urine and in serum and of human placental lactogen hormone in serum in the last part of pregnancy Am J Obstet Gynecol 126 834 1976)

Fetal Reserve

The oxytocin challenge test (OCT) was designed to test the reaction of the fetus to the stress of uterine contractions The patient should be placed on her side or propped in a sitting position and an external (Doppler) fetal monitor applied If spontaneous uterine contractions are not apparent, oxytocin should be administered by infusion pump until regular (3–4 every 10 min) contractions are observed

INTERPRETATION

Positive Repetitive late decelerations in association with normal contractions (small decelerations may be significant and are often very difficult to see)
Negative No late decelerations observed
Suspicious Occasional late or unclassified decelerations
Unsatisfactory Poor quality recording

There is general agreement that a negative test is a reliable indicator of a fetus in good condition The significance of a positive test however is uncertain A false positive rate as high as 50% has been reported

During study of the OCT, a number of investigators noted the appearance of periodic accelerations of the fetal heart, which were associated with fetal movements or uterine contractions and occurred in healthy fetuses These observations led to the development of the *non stress test* also called fetal activity determination (FAD) test A healthy fetus has frequent movements, and frequent cardiac acceleration patterns are noted This test, though still under investigation, shows great promise and is a much more acceptable screening procedure than the OCT

Since no single test of fetal well-being is 100% accurate, it is best to use two or three, preferably measuring different aspects of the fetal status A positive OCT alone may or may not be significant, but a positive OCT in the presence of falling estriols is highly significant Similarly, a slowdown in growth of the biparietal diameter becomes much more ominous when accompanied by a drop in estriol excretion or a positive OCT.

However, test results may give a confusing and contradictory picture of the fetal status, making the clinician feel a victim of his own technology The simple method of management outlined earlier has much to recommend it For this, the patient with toxemia is hospitalized for the remainder of her pregnancy If the disease deteriorates or clears and then recurs during the bed rest regimen, the situation may be taken as evidence that the fetoplacental unit is failing, the patient should then be delivered The results in terms of both maternal and fetal outcome are excellent, and the cost of hospitalization may well be offset by the savings on batteries of sophisticated tests

If prolongation of pregnancy is thought to be hazardous to the fetus, the maturity of the fetus should be estimated There are a number of tests available to assess maturity (Table 6-3) Of these, the most important is the lecithin/sphingomyelin (L/S) ratio An L/S ratio under 1 5/1 indicates that the fetus runs a substantial risk of developing severe respiratory distress syndrome (RDS) if delivered A ratio of 1 5–2 0/1 indicates a risk of mild RDS while a ratio of 2 0/1 or greater implies little risk of RDS The test, however, is time consuming, and more rapid methods are being continually sought (Sbarra *et al*, 1976) The use of steroids to accelerate lung maturity shows promise

TABLE 6-3 Assessment of Fetal Maturity

Test	Significance	Comment
LMP	Nageles rule	
Quickening	20–22 wk (primigravida) 16–20 wk (multipara)	Reliable information may not be available
Fetal heart tones first heard	20 wk	
X ray films for epiphyses	*Lower femoral at 36–38 wk* Upper tibial at 40 wk	*Negative results do* not exclude maturity
Serial ultra sonic cephalometry	Measures growth of fetal head	Must be started at 20–30 wk
Amniotic fluid		
Fat cells	>10% fat cells indicates skin maturity	Conflicting evidence of accuracy
Creatinine	>1 8 mg/100 ml indicates renal (and muscle) maturity	20–30% inaccuracy in toxemia renal disease and sickle-cell disease
Bilirubin	Falls to zero with maturing of liver (36 wk)	Large spread in values false positives occur
L/S ratio	2 1 indicates lung maturity	Best available but 6% inaccuracy in complicated pregnancy

OBSTETRIC DISPOSITION

As stated earlier, the patient with severe eclamptogenic toxemia who does not respond to medical treatment should be delivered, regardless of fetal maturity.

TIMING OF DELIVERY

In cases of severe eclamptogenic toxemia and eclampsia, it is usual to stabilize the patient for 24 hours and then to deliver her. However, in fulminating cases or in patients who do not respond to attempts at stabilization, the decision to deliver may be made after 12 hours or even earlier.

In mild to moderate cases, the decision will often be made when signs of fetal deterioration occur. During the last few weeks of pregnancy in such patients, there should be no hesitation in delivering the infant if there has been no improvement in maternal and/or fetal status after bed rest in the hospital.

MODE OF DELIVERY

Most patients with eclamptogenic toxemia respond readily to attempts to induce labor, even when the cervix appears unfavorable. Unless there is a contraindication (e.g., breech presentation), labor should be induced by artificial rupture of the membranes followed by oxytocin infusion. In severe cases, it is particularly important to remember that the patient needs to be *delivered* promptly, not just induced. Therefore, day after day of oxytocin induction without artificial rupture of the membranes is not recommended.

Fetal monitoring should be continued throughout the induction and labor period, and evidence of fetal compromise should be treated promptly by cesarean section, if necessary. Similarly, failure of induction should be followed promptly by cesarean section. Fulminating toxemia may first manifest itself during induction or labor, and if it cannot readily be managed by medical treatment, the pregnancy should be terminated by cesarean section.

CARE OF THE PATIENT DURING LABOR

The patient should be kept adequately hydrated and sedated, and both mother and fetus should be continuously monitored for any evidence of deterioration in condition. Adequate analgesia is important, and narcotics and regional anesthesia may both be used. Epidural and spinal anesthesia should not be used in patients with toxemia that is superimposed on chronic hypertension, but epidural anesthesia has been used in "pure" toxemia (Nicholas, 1975).

The second stage of labor is best completed by elective outlet forceps. Ergonovine (Methergine) should be avoided in the management of the third stage of labor because it may exacerbate the hypertension.

ECLAMPSIA

Once a patient has suffered convulsions, management consists of preventing further seizures, stabilization of the underlying toxemia, and termination of the pregnancy at the appropriate time. In addition, cardiorespiratory problems must be dealt with promptly and adequately.

MANAGEMENT OF THE ECLAMPTIC SEIZURE

1. Place the patient in a bed with siderails and keep her on her side Use gentle restraint, and loosen tight clothing
2. Prevent tongue biting by means of a mouth gag or padded tongue-blade
3. Administer oxygen
4. Give sodium amobarbital, 0 25–0 5 g, slowly intravenously DO NOT OVERSEDATE the patient

GENERAL MANAGEMENT

1. Keep the patient in a quiet, darkened room under constant observation
2. Manage the underlying toxemia as described earlier in this chapter
3. Fluids

   Limit the fluid intake per 24 hours to 1000 ml plus the amount of urinary output over the preceding 24 hours unless the patient is hypovolemic
4. Routine observations
   - A. Check blood pressure, pulse, and respiratory rate at least every 15 min
   - B. Check the lungs frequently, as acute pulmonary edema is common in eclamptic patients
   - C. Keep a careful intake-output chart
   - D. Obtain blood for typing, crossmatching, and for a base-line hematocrit. Hemoconcentration is common in patients with eclamptogenic vasospastic toxemia Determine serum protein by electrophoresis serum electrolytes, blood uric acid, and blood urea nitrogen
   - E. Monitor fetal heart and uterine contractions
   - F. By means of an in dwelling catheter, have urine collections made over each 24-hour period for protein and urinary estriol determinations, until labor is induced
5. Cardiorespiratory complications

   Anticipate these so they may be promptly treated
   - A. Consider prophylactic digitalization with 1 6 mg lanatoside C intravenously if the pulse rate is over 120/min and there is no contraindication such as hypokalemia Thereafter, administer 0 25–0 5 mg digoxin daily, to maintain digitalization Obtain an electrocardiogram prior to digitalization
   - B. Treat acute pulmonary edema as described earlier in this chapter
   - C. Provide respiratory assistance by endotracheal tube if necessary because of gross edema or tongue biting Tracheostomy may occasionally be necessary
6. Disseminated intravascular coagulation

   This is rarely a serious problem The presence of hematuria should raise suspicion but is more commonly due to trauma from the Foley catheter If hematuria becomes marked, the diagnosis should be established by a coagulation profile showing thrombocytopenia, with depression of fibrinogen and fibrinogen factors (V, VIII, and XIII), and the presence of fibrin monomers and fibrin/fibrinogen degradation products Then the use of heparin should be considered (see Ch 3, Shock)

OBSTETRIC DISPOSITION

The obstetric disposition is the same as that presented earlier for preeclamptic toxemia

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Chapter 7

Hemorrhage in Early Pregnancy

Ralph E. Woods, Denis Cavanagh

"If you do not know a thing you are quite sure not to suspect it and if you do not suspect a thing you are almost certain not to find it"
Matthews Duncan
Medical Times and Gazette 1878 p 729

Hemorrhage in early pregnancy may be caused by a pedunculated fibroid, cervical mucus polyp, cervical erosion, pregnancy in a bicornuate uterus, or vulvar and vaginal lesions More important, however, from the standpoint of obstetric emergencies are abortion, ectopic pregnancy, trophoblastic disease, cervical carcinoma and chemical ulceration of the vagina (Fig 7-1)

The patient may present in shock due to severe intraperitoneal or external bleeding In such situations, there will be little difficulty in recognizing the serious nature of her problem Often however, bleeding is minimal It may be relatively benign or may be early evidence of a life threatening situation Recognition may be difficult Particularly in these cases, systematic patient evaluation is essential for early diagnosis

PATIENT EVALUATION

Clinical methods remain the mainstay of patient evaluation Supplementary studies may be necessary, but a careful history and complete physical examination are often crucial in defining the nature of the problem Several examinations may be necessary before a diagnosis can be made Often, during this period of evaluation, the problem will become acute and patently obvious

HISTORY

The time frame in which the patient's symptoms have developed should be obtained The basis for comparison as to normal/abnormal is her usual menstrual pattern. It is informative to know the date of onset of the last menstrual period, its duration, the approximate amount of flow, the usual cycle pat-

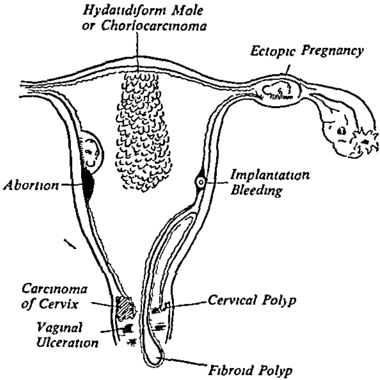


FIG 7-1 Causes of bleeding in early pregnancy

tern, and the breast and pelvic symptoms that ordinarily accompany her menstrual period Information should be sought as to whether she has been using basal body temperature or cervical mucus patterns to regulate coital activity

Knowledge of contraceptive practices (particularly the use of an intrauterine contraceptive device), of coital partners, previous pelvic inflammatory disease (including postabortal or postpregnancy), and previous pregnancy interruptions may be helpful Information about infertility, infertility studies, and lower abdominal surgery is also important.

EXAMINATION

Vital Signs

Temperature, pulse, respiration, and blood pressure should all be checked

Abdomen

The patient's abdomen must be evaluated carefully for point tenderness, voluntary guarding, rigidity, rebound tenderness, distension, and reduced bowel sounds The presence of any of these may indicate peritoneal irritation due to early intraperitoneal bleeding

Pelvis

BIMANUAL EXAMINATION Manual examination should be gentle If movement of the cervix laterally elicits pain on the same side toward which the cervix is moved this suggests adnexal pathology So far as palpable adnexal masses are concerned, it should be remembered that an enlarged and tender corpus luteum is frequently present in early pregnancy Attention must be paid to uterine size, consistency, shape, and location especially in regard to whether an intrauterine pregnancy is actually present or not A retroverted uterus may cause confusion, and pain caused by attempts to antevert it may be significant A bicornuate uterus or other anomaly may be detected The uterus itself may be surprisingly soft in early pregnancy However, when decidual hemorrhage is present, the uterus may be tender and irritable and may contract as it is palpated The classic clinical signs of pregnancy may be evident 1) Hegar's softening of the supravaginal portion of the cervix, 2) Piskacek's sign—asymmetry with softening of the uterus in the area of implantation

SPECULUM EXAMINATION Cervical lesions may be evident A double cervix may be seen Papanicolaou smears should be taken Cervical cyanosis (Chadwick's sign) may be evident Ordinarily, its presence suggests pregnancy, but it is of limited value in the presence of bleeding The area of vagina posterior to the cervix should be carefully inspected for bulging of the cul de sac

LABORATORY TESTS

Blood Tests

A complete blood count, including differential white blood cell count, and blood type should be obtained A patient may lose a considerable amount of blood before she shows signs of iron deficiency anemia or shock If she is starting into a series of problems with diminished iron reserves, or a low hemoglobin level, she may be less able to tolerate acute blood loss or anesthesia if it should become necessary The information is valuable

Pregnancy Tests

Biologic pregnancy tests have largely been replaced by immunologic assays Three types are generally available 1) Latex agglutination inhibition—slide tests, *e g*, in the United States UCG, Pregnosticon, Gravindex, 2) Latex agglutination test, *e g*, in the United States DAP, 3) Hemagglutination inhibition tube tests, *e g*, in the United States Pregnosticon, UCG, Accusphere All depend on the level of ouput of chorionic gonadotropin by trophoblastic tissue This is a function of trophoblastic viability and the length of gestation It has been shown that between day 24 and day 40 after the first day of the last menstrual period, the urinary excretion of chorionic gonadotropin ranges between 1000 and 2000 IU/24 hours The tube hemagglutination inhibition tests are the most sensitive of the three types, but require about 2 hours to perform They will detect gonadotropin levels as low as 750 and 1000 IU/24 hours Thus, they will detect the presence of living trophoblastic tissue early The slide tests are quicker but less sensitive (range 1000–2000 IU/24 hours)

These tests are helpful but they may have to be repeated sequentially If the patient is taking phenothiazines, promethazine, phenobarbital, or aspirin, interference with the reaction is possible Since the reagents crossreact with pituitary gonadotropin (LH), the sensitive tests may give false-positive results when pituitary gonadotropin excretion levels are high Because of these possibilities, care must be used in interpreting the results

More recently, a radioimmunoassay procedure for the detection of the beta subunit of human chorionic gonadotropin has been reported This is the only technique that differentiates between HCG and LH in specimens in which the amount is so low that it could be accounted for by either source of production It is now recognized that in patients with trophoblastic disease, the disease may persist with excretion of gonadotropin below the sensitivity level of the usual immunoassay procedures (Delfs 1975) It has been recommended that patients with this disease be followed with the radioimmunoassay procedure for detection of the beta subunit If the procedure is not available locally, serum can be sent to one of the regional trophoblastic centers for assay, thus assuring better follow up of these patients (Figs 7-2, 7-3)

SONAR

The use of pulsed high frequency ultrasound for medical diagnosis has expanded rapidly in recent years Development of this technique (medical sonar) has been largely due to the enthusiastic and determined perseverance of physicians, physicists, and engineers New interest has been stimulated recently by the development of an effective "gray scale' and 'real-time' imaging Donald has reviewed the several stages of development and acceptance that this modality has undergone

There has always been concern over possible long term side effects of medical sonar Pulsed echo sounding utilizes a form of physical mechanical energy at levels well below those used in power ultrasound To date there has been no validated evidence of tissue damage, even in embryonic tissue Nonetheless, it is possible that a safety threshold exists for this form of energy The procedure appears to be safe, but until the matter is finally settled one or two generations from now, it behooves sonar users to minimize energy usage

Sonar has influenced the practice of obstetrics in a number of ways It has not replaced clinical judgment but it is a material aid in the diagnosis of the causes of vaginal bleeding both in early and late pregnancy A scan will show only what is present at the time of the examination The durations of gestation at which various structural features make their appearance are presented in Table 7-1 Thus the examination may have to be repeated one or more times to verify the diagnosis

When sonar is used carefully, there is no doubt as to its effectiveness in demonstrating 1) intrauterine pregnancy when ectopic pregnancy is suspected, 2) trophoblastic disease, 3) the 'threatened' abortion that will abort because it is a blighted ovum, 4) the "threatened" abortion which is unlikely to abort, 5) the pregnancy in a bicornuate uterus, 6) the missed abortion early as well as late, 7) placental separation in early pregnancy, 8) the early intrauterine gestation in conjunction with the intrauterine contraceptive device (Figs 7-4

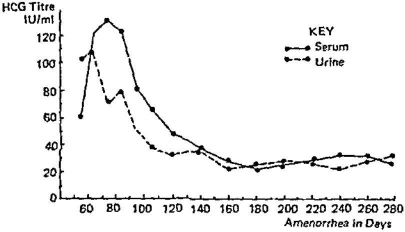


FIG 7-2 Mean serum and urine chorionic gonadotropin levels in 600 normally pregnant women, the technique of hemagglutination inhibition was used (Teoh ES J Obstet Gynaecol Br Commonw 74 77, 1967)

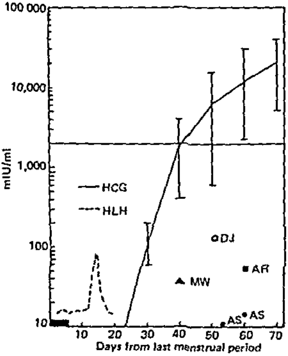


FIG 7-3 Rising mean serum HCG levels of 15 patients with normally developing intrauterine pregnancies Brackets outline extent of scatter *Initials correspond to serum HCG levels of the 4 patients with ectopic pregnancies* Horizontal line at 2000 *mIU/ml is approximate* area where most immunologic pregnancy tests become positive (Kosasa TS et al Obstet Gynecol 42 268, 1973)

through 7-16) In later pregnancy, the abnormally located placenta, and certain placental accidents can also be demonstrated.

Some equipment is capable of demonstrating fetal heart movement from very early in pregnancy (ninth week after the onset of the last menstrual period) This provides added perspective because fetal viability can be monitored before the time when fetal heart tones would ordinarily be heard

CULDOCENTESIS

The value of aspiration of fluid from the posterior cul-de-sac as a means of diagnosing intraabdominal disease has been amply documented (Table 7-2) When nonclotting blood is obtained, it may signify either a ruptured corpus luteum or a ruptured ectopic pregnancy A positive tap is an indication for laparotomy However a negative tap does not exclude an ectopic pregnancy

The procedure is simple and safe It can be done as an outpatient procedure The recommended technique is as follows The patient sits up for 5-10 min to allow blood (or fluid) to gravitate to the cul-de-sac She is then put in lithotomy position If she is uncomfortable, meperidine (Demerol), 25-50 mg, may be given intravenously, slowly No local anesthetic is used (Fig 7-17)

1. A large vaginal speculum is inserted The vagina is gently cleansed with an antiseptic solution, such as povidone-iodine (Betadine)
2. A tenaculum is placed on the posterior lip of the cervix
3. The cervix is elevated The posterior fornix is exposed and cleansed with the antiseptic solution Excess solution is wiped away with sterile gauze
4. A number 10-gauge spinal needle with a syringe attached is inserted at a point 1 cm below the junction of the cervix and vaginal mucosa
5. The needle insertion should be quick, and to a depth of about 2 cm Suction is applied as the needle is withdrawn
6. If nonclotting blood is obtained, the diagnosis of intraperitoneal bleeding is established If no blood or fluid is obtained, the needle point may be above the cul-de-sac pool, so, as it is withdrawn slowly, intermittent suction should be maintained If the tap is dry, it should be repeated at least once If blood is aspirated but clots promptly, this suggests a "traumatic tap," the needle usually having been inserted into the posterior wall of a retroposed uterus

TABLE 7-1 Sonographic Changes in Early Pregnancy

Time since LMP (wk)	Sonographic changes
5-6	Gestational sac becomes evident (evident until 13 wk)
6-7	Embryonic tissue evident
9	Fetal heart pattern demonstrable
9	Placenta becomes identifiable
11-12	Fetal head becomes identifiable

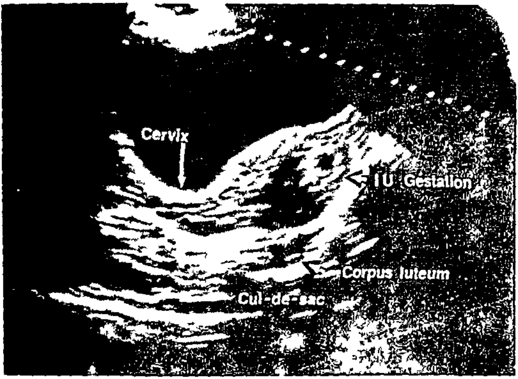
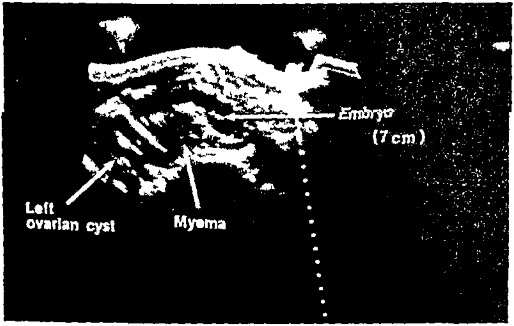


FIG 7-4 Sonogram (Saggital section) Suspected ectopic pregnancy Bloody fluid from cul-de sac aspiration Sonar shows intrauterine gestation sac in fundal area of the uterus with a cyst (corpus luteum) behind the uterus

FIG 7 5 Sonogram (Transverse section) Early intrauterine pregnancy with myoma underlying the placental s te and a left ovarian cyst (corpus luteum)



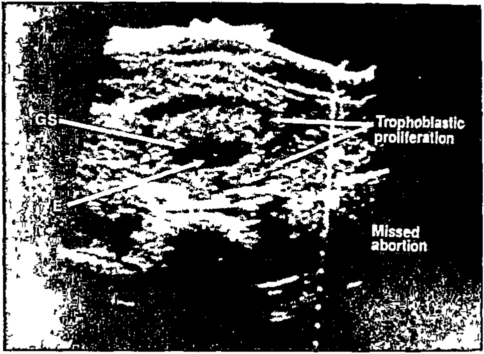
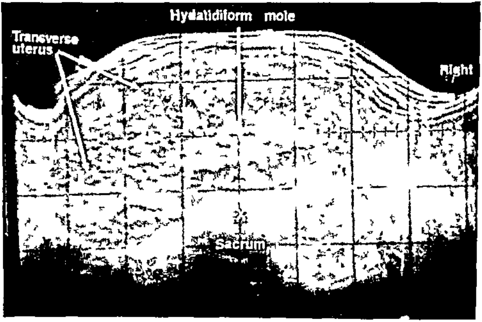


FIG 7 6 Sonogram (Transverse section) Missed abortion Positive pregnancy test. Sonar shows degenerating embryo with marked trophoblastic overgrowth GS = Gestation Sac E = Embryo

FIG 7 7 Sonogram (Transverse section) Classic hydatidiform mole



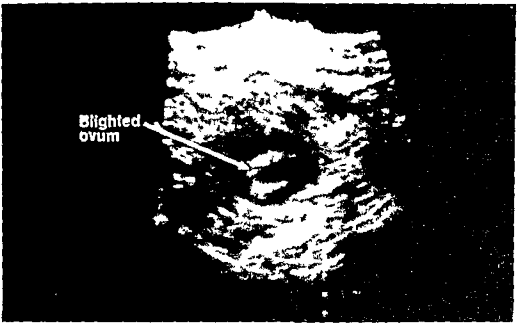
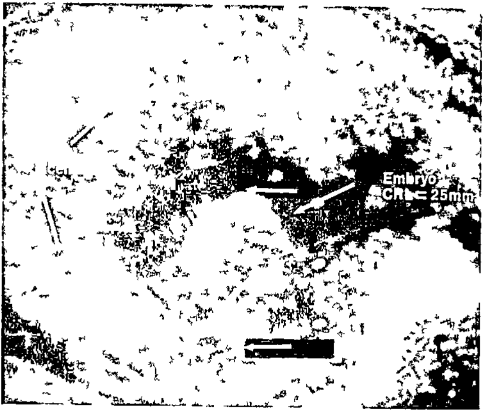
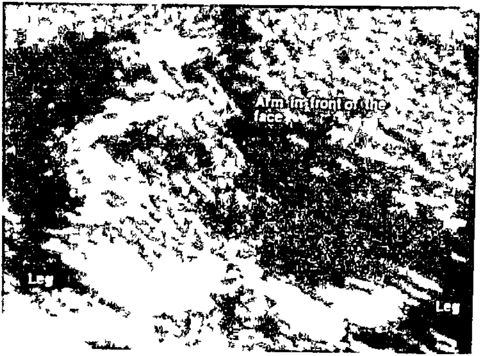


FIG 7 8 Sonogram (Transverse section) Inevitable abortion—blighted ovum with broken gestational sac

FIG 7 9 Sonogram (Saggital section) Twin anembryonic gestational sacs showing degeneration of wall

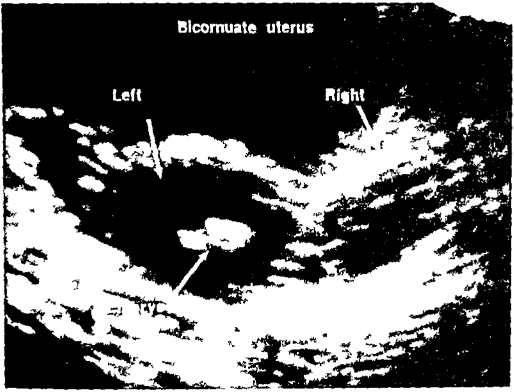

Embryo
CRL = 25mm

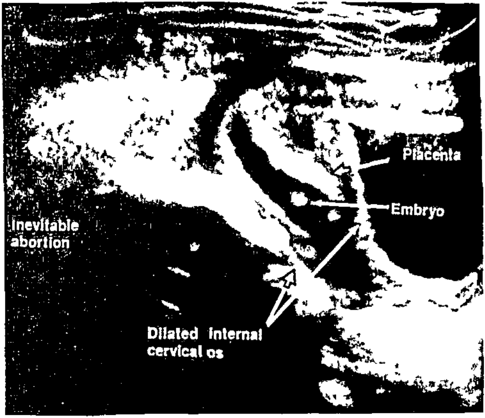

Arm in front of the face
Leg
Leg

◀ FIG 7 10 Sonogram (Transverse section) Threatened abortion repeated cramping and spotting in first 6 weeks after last menstrual period Embryo shows crown to rump length of 25 mm compatible with a normal gestation of 9 weeks since the last menstrual period

◀ FIG 7 11 Sonogram (Saggital section) Same fetus as in 7 10 but now 12 weeks since last menstrual period

FIG 7 12. Sonogram (Transverse section) Early pregnancy in bicornuate uterus associated with spotting from the non-pregnant horn (An unruptured ectop c pregnancy m ght show a similar picture)

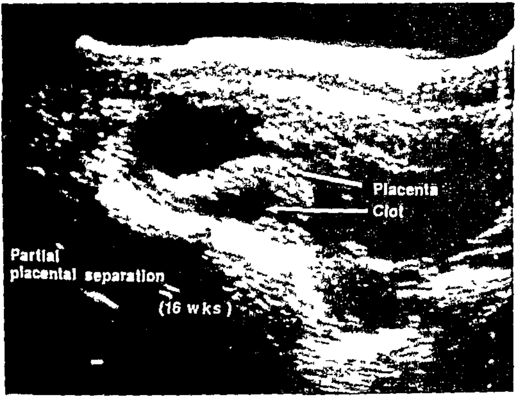



Placenta
Embryo
Inevitable
abortion
Dilated internal
cervical os

◀ FIG 7-13 Sonogram (Saggital section) Inevitable abortion Blighted embryonic tissue with dilated cervical os and canal, but no bleeding as yet.

◀ FIG 7-14 *Sonogram (Transverse section) Missed abortion Crown to rump length (CRL) at 20 mm for 70 days of amenorrhea (CRL standards from Robinson H, Fleming JF Br J Obstet Gynaecol 82 702, 1975)*

FIG 7 15 Sonogram (Saggital view) Early hemorrhage at 16 weeks after last menstrual period due to partial separation of placenta Patient delivered a healthy infant at term



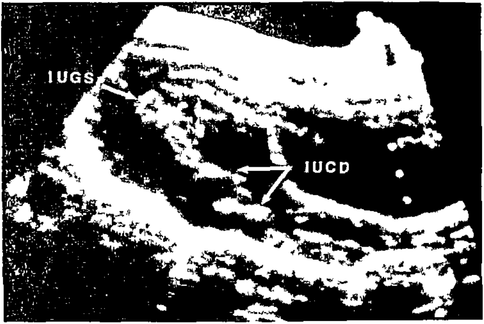


FIG 7 16 Sonogram (Saggital section) Threatened abortion Intrauterine gestational sac (IUGS) in fundus with intrauterine contraception device (IUCD) in lower portion of uterus

SURGICAL PROCEDURES

Laparoscopy

This procedure is useful in clarifying the cause of pelvic pain, particularly when an unruptured ectopic pregnancy is suspected It is a useful measure in the diagnosis of intraabdominal disease

Culdoscopy

This procedure has largely been replaced by laparoscopy

Posterior Colpotomy

Through this incision, the pelvis can be explored digitally, and limited operative treatment can be accomplished

Laparotomy

This is the procedure of choice if there is evidence of significant intraperitoneal bleeding. A vertical midline incision should be used, so that the upper abdomen can be explored adequately, if the bleeding source is not found in the pelvis

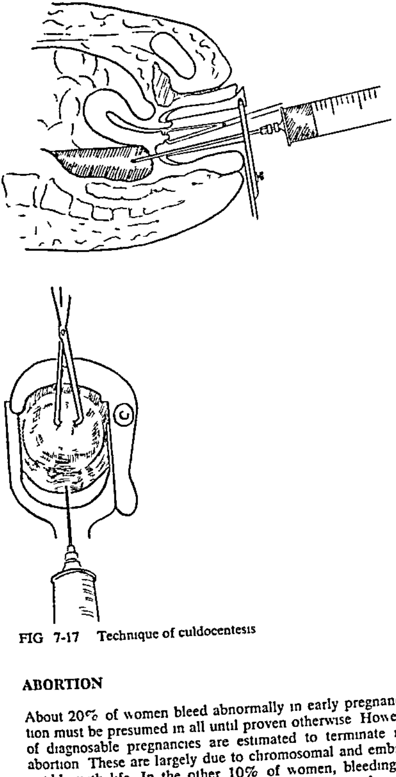


FIG 7-17 Technique of culdocentesis

ABORTION

About 20% of women bleed abnormally in early pregnancy Threatened abortion must be presumed in all until proven otherwise However, only about 10% of diagnosable pregnancies are estimated to terminate in early spontaneous abortion These are largely due to chromosomal and embryonic defects incompatible with life In the other 10% of women, bleeding could be caused by threatened abortion or the development of placental problems, but it is not

TABLE 7-2 The Value of Culdocentesis in the Diagnosis of Intraabdominal Diseases

Description of fluid aspirated	Possible cause	Recommendations
Bright or dark red nonclotting blood small clots on smear	Intraabdominal bleeding due to ruptured ectopic pregnancy bleeding corpus luteum cyst or other cause	Laparotomy
Old brown nonclotting blood with clot particles on smear	Old ruptured ectopic old pelvic hematocele	Examination under anesthesia colpotomy and/or laparotomy
Fresh blood clotting withim 10 min	Traumatic tap with aspiration of blood from pelvic blood vessel	Repeat culdocentesis if same fluid aspirated treat as in second item
Blood tinged serous fluid	Ovulatory bleeding ruptured ovarian cyst traumatic tap	Repeat culdocentesis if same fluid aspirated treat as in second item
Clear straw colored serous fluid in small amount	Normal peritoneal fluid unruptured ectopic pregnancy still to be considered if menstrual upset lower abdominal pain and/or adnexal mass	Repeat culdocentesis if same fluid aspirated treat as in second item
Clear yellow fluid sometimes clotting	Hydrosalpinx encysted peritoneal fluid or ovarian cyst content where ovarian malignancy is suspected culdocentesis should be avoided because of danger of disseminating disease	Colpotomy with great care laparotomy preferred if ovarian malignancy is suspected
Turbid amber or dark yellow fluid in moderate amount with bacteria on Gram stained smear	Acute salpingitis rule out appendicitis	Laparotomy if any suspicion of appendiceal lesion

Frank pus	Pelvic abscess	Incision and drainage through cul-de-sac if abscess dissecting rectovaginal septum
Fecal material, soft or of various degrees of fluidity	Traumatic rectal perforation, rule out closed loop obstruction by history, abdominal examination and x-ray film of abdomen	Repeat tap, usually no morbidity but observe for peritonitis
Cloudy, acid, sour-smelling fluid containing food particles and mucin	Perforation of gastric or duodenal ulcer (characteristically upper abdominal pain)	Laparotomy
Bile stained fluid, cholesterol crystals seen microscopically	Ruptured gallbladder (characteristically upper abdominal pain)	Laparotomy
Oily "beef-juice' fluid, microscopic examination shows many polymorphs but few bacteria, high amylase content	Acute pancreatitis (characteristically upper abdominal pain)	If diagnosis confirmed by clinical picture and serum amylase estimation, treat medically
Urine	Ruptured urinary bladder	Laparotomy
Nonproductive	This is NOT a negative tap, rather consider that cul-de-sac has not been entered	Repeat culdocentesis; substitute abdominal paracentesis if indicated, if ectopic pregnancy still suspected treat as in second item

(Cavanagh D Obstetrical Emergencies, 1st ed Springfield CC Thomas, 1961)

necessarily incompatible with a successful pregnancy Carefully performed serial sonar examinations have been very helpful in identifying the patient who will abort (blighted ovum)

Diagnosis and management are altered by this approach If no embryonic tissue is seen to develop within the gestational sac, or if growth ceases, or if the fetal heart patterns disappear after having been previously demonstrated, then the evidence is overwhelming that the fetus is dead or dying Later in pregnancy, bleeding caused by placental problems can be identified in a large number of cases Cessation of fetal growth will be evident by sonar long before it is clinically evident Because fetal life and growth can be monitored sequentially, individualization of therapy is possible

When this approach is not possible because of the urgency of the problem, traditional approaches must be utilized The subcategories of abortion are as follows threatened, inevitable, incomplete, complete, missed, and septic These and the features upon which the diagnosis is based are presented in Table 7-3

THREATENED ABORTION

It is customary to advise restriction of general and sexual activity, at least while bleeding is evident, but in view of the report of Hertig and Livingston (1944) that in spontaneous abortion an abnormal conceptus is present in most cases, the patient should generally be allowed up 48 hours after the acute episode A pelvic examination must be made to try to rule out the possibility of an ectopic pregnancy Trophoblastic disease must also be considered Hemoglobin, hematocrit, and HCG determinations are advisable The patient should be cautioned to report any increase in bleeding, development of cramping, or passage of tissue Tissue should be saved for examination Progestational agents should not be used except in cases where a demonstrable luteal deficiency is present, otherwise blighted ova may be retained Folic acid (300 μg/day) and vitamin B_6 might be considered, in view of the large demand by the developing trophoblast Should an intrauterine contraceptive device be in situ and the string be accessible the device should be removed If the history indicates the use of one and one is demonstrated by x-ray film or sonogram to be lying high in the uterus, no attempt at removal should be made unless severe endometritis is present

In threatened abortion, bleeding is usually light but may become very heavy, a sign that the process has become inevitable When bleeding is heavy, and yet the cervix is closed, careful judgment is necessary If the patient's life is endangered by the heavy or protracted bleeding, arrest of hemorrhage may be accomplished by the use of an oxytocin infusion If after a period of several hours, this is ineffective, surgical evacuation may be necessary

INEVITABLE ABORTION

When the internal os becomes dilated the process of abortion is described as inevitable This may occur at any time during early pregnancy The onset may be signalled by 1) development of uterine cramping, 2) heavy vaginal bleeding, or 3) a gush of fluid as a result of rupture of the amniotic sac Hospitalization is necessary, especially if the duration of pregnancy has gone beyond the 12th

TABLE 7-3. Clinical Diagnosis of Abortion

Type abortion	Fever	Complaint of abdominal cramps	Bleeding (amt)	Tissue passed vaginally	On examination: Tissue in vagina	On examination: Status of internal cervical os	On examination: Uterine size
Threatened	None	Slight	Slight	None	None	Closed	Commensurate with "dates"
Inevitable	None	Moderate	Moderate	None	None	Open with membranes or tissue bulging	Commensurate with "dates"
Incomplete	None	Severe	Severe	Placental or fetal tissue	Possibly	Open with tissue in cervical canal	Smaller than "dates"
Complete	None	None	Minimal	Complete placenta and fetus	Possibily	Closed with no tissue in cervical canal	Smaller than "dates"
Missed	None	No cramps no life felt, no FHT (Doptone)	Brownish discharge, heavy if DIC	None	None	Closed	Smaller than expected and not enlarging
Septic (usually criminal)	Yes	Usually severe and out of proportion to bleeding	Slight to severe	Possibly	Possibly typically have foul discharge and parametrial tenderness	Closed or open and with or without tissue in canal	Commensurate or smaller, depending on stage of abortion reached, uterus is tender

(Revised from Cavanagh D Obstetrical Emergencies, 1st ed Springfield, CC Thomas, 1961)

week After careful pelvic assessment, and base-line determinations of hemoglobin, hematocrit, white blood cell count, differential count, Rh type, and major blood group determination, treatment is instituted If the woman's condition is stable and uterine contractions are evident she may be given sedatives such as diazepam (Valium), 5 mg IM or analgesics such as meperedine hydrochloride (Demerol) 50–100 mg IM, as needed

The duration of gestation and size of the uterus will determine the methods chosen to effect expulsion Early in pregnancy, dilatation of the cervix with Hegars or Hanks dilators and evacuation of the uterine cavity with ring forceps and standard or suction curets are indicated An oxytocin infusion must be running Care must be used not to perforate the uterus Perforation with the suction curet is potentially more dangerous than with the standard curet If either occurs, the patient must be watched carefully for the development of intraperitoneal hemorrhage or infection Aspiration of a loop of bowel into the suction curet is an indication for immediate laparotomy

Later in pregnancy, between the 12th and 20th weeks, oxytocin (10–20 units in 1000 ml 5% dextrose in water) should be infused initially, and evacuation is deferred until the uterus has decreased in size As a general rule, evacuation should not be attempted *per vaginam* if the uterus is larger than 14 weeks' size On rare occasions hysterotomy and even hysterectomy may be necessary, because of the degree of hemorrhage, infection, or other complications that may develop Evacuation should be performed under general anesthesia but if this is not available, or is contraindicated, the procedure can be readily performed under paracervical block Following the procedure, methyl ergonovine (Methergine), 0 2 mg IM should be given every 8 hours for 3 doses in addition to continuing the oxytocin infusion for 6–8 hours Hemorrhagic shock must be combated with properly matched packed red cell or whole blood transfusions

INCOMPLETE AND COMPLETE ABORTIONS

Management involves an estimate of whether residual trophoblastic tissue is present after some expulsion of tissue has occurred In many cases, the process seems complete, because after expulsion of tissue, bleeding stops If the uterus is small, firm, and remains well contracted there is probably no need for a D&C With a gestational duration of more than the 40 days, there is more likely to be residual tissue A D&C is advisable even if active bleeding is not present at the time of examination, but not after 14 weeks Ergonovine (Methergine), 0 2 mg IM may be given while awaiting the D&C However, oxytocin (Pitocin) should preferably be given by infusion in a dose of 10 units per 1000 ml dextrose in water Postabortal sonar examination through a full urinary bladder may be helpful in demonstrating retained tissue (see Fig 11-8)

MISSED ABORTION

By definition, missed abortion is the retention for 4 weeks or more of the products of conception after the death of the embryo or fetus Most missed abortions terminate spontaneously within 4–5 weeks after fetal death A major complication is the development of disseminated intravascular coagulation (DIC), also known as intravascular coagulation fibrinolysis (ICF) Provided

the patient can cope with the knowledge that there is a dead fetus within her, she can be monitored by weekly fibrinogen determinations starting 4 weeks after the presumed death of the fetus Disseminated intravascular coagulation is rarely seen before this time but may occur sooner and be worse if Rh isoimmunization is involved If the fibrinogen level falls below 150 mg/100 ml, or if the patient develops spontaneous ecchymoses, gum bleeding, epistaxis, or the like, intervention becomes necessary Any of the previously mentioned techniques may be used

Prostaglandins might be more effective than oxytocin in effecting expulsion Gastrointestinal side effects are prominent but may be alleviated by pretreatment with antiemetics such as prochlorperazine (Compazine), 5–10 mg IM, and antidiarrheal agents such as diphenoxylate HCl (Lomotil), 5 mg orally Presently recommended techniques include 1) intraamniotic instillation of prostaglandin $F_{2}\alpha$, in 40 mg doses Simultaneous administration of dilute oxytocin (5 units/liter) may facilitate the effect, 2) intramuscular injections of 250 μg (15S)-15 methyl prostaglandin $F_{2}\alpha$ every 3 hours until expulsion is complete Prostaglandin vaginal inserts are also available The use of amniotomy or laminaria tests may lead to sepsis, if they must be used the administration of a broad spectrum antibiotic is advisable The management of DIC (ICF) in this situation—the dead fetus syndrome—is discussed in Chapter 2

SEPTIC ABORTION

The problems related to septic abortion are discussed in Chapters 3 and 4

TISSUE EXAMINATION

It is strongly advised that all tissue obtained, particularly in early pregnancy, be examined for evidence of trophoblastic disease or as definitive confirmation of intrauterine pregnancy The tissue expelled or curetted may prove to be a decidual cast without chorionic villi, or it may show a definite Arias-Stella reaction Either of these findings would suggest the need for laparoscopy to rule out ectopic pregnancy

ECTOPIC PREGNANCY

Ectopic pregnancy was the third most common cause of maternal death in the United States in 1975 Reported incidences vary between 1 in 90 to 1 in 200 pregnancies The basic cause is impaired passage of the fertilized ovum through the tube There is general agreement that since the introduction of antibiotics for the treatment of pelvic inflammatory disease, the incidence of ectopic pregnancies has climbed Although endosalpingitis is considered to be the most common cause of partial tubal obstruction, there is often no gross evidence or history of pelvic infection There is continued uncertainty as to whether the intrauterine contraceptive device (IUCD) has caused an increase in incidence or whether ectopic pregnancies are more noticeable because intrauterine pregnancies have declined so markedly In 10–15% of the cases, the corpus luteum is in the contralateral ovary Iffy (1963) has long contended that late-

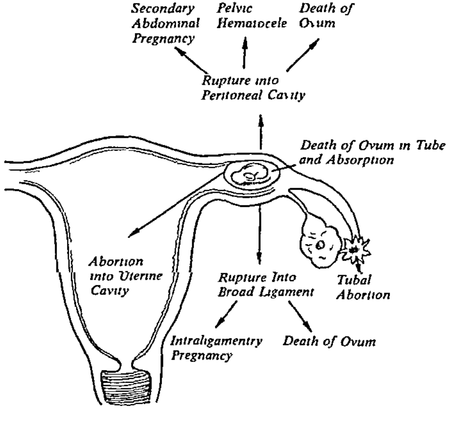
cycle conception, before the last "normal" menstrual period, is a major contributing cause

There are numerous types of ectopic pregnancy, but the most common are tubal pregnancies Nidation occurs principally in the ampullary, isthmic, fimbrial, or interstitial segments of the tube in that order of frequency

Within the tube, conditions for nidation are poor The penetrating trophoblast burrows into the muscularis of the tube and ultimately penetrates an arteriole or artery Hemorrhage into and under the implantation site may then dissect the gestational sac from its attachments It may die and be resorbed or it may be aborted in retrograde fashion through the tube into the peritoneal cavity The rapidity with which this tubal abortion occurs and the amount of blood expelled influence the acuteness of the symptoms Alternatively, if trophoblastic attachments persist, some degree of growth will occur Eventually, villus penetration and the surrounding hemorrhage will lead to a rupture of the tubal wall and extrusion of blood and embryonic tissue into the peritoneal cavity or between the leaves of the broad ligament (Fig 7-18)

The time after fertilization when these events occur varies greatly Since implantation usually begins by the seventh day after fertilization, the patient may become symptomatic before or about the time she would expect a menstrual

FIG 7 18 Fate of the ovum in tubal pregnancy



period More often, the process continues for 6–9 weeks before becoming clinically evident

DIAGNOSIS

Usually there is some degree of menstrual delay, followed by slight but persistent vaginal bleeding due to breakdown of the decidua This may confuse the clinical picture by suggesting a threatened abortion, particularly if, after a period of uterine cramping, a decidual cast is passed, or it may be mistaken for a normal period Pain is present in over 90% of cases but varies in location and type It is usually diffuse over the lower abdomen but may be unilateral It may be continuous or intermittent and cramping in nature, or even acute and sharp

Symptoms and findings depend on the type of ectopic pregnancy that exists The site, amount of bleeding, and the rapidity with which the process develops will affect the clinical picture Two patterns have been described The chronic pattern develops over a period of weeks and is associated with repeated small episodes of bleeding These are accompanied by repeated attacks of pain, vaginal spotting, some degree of uterine enlargement, and the development of a pelvic mass Eventually severe hemorrhage with intraperitoneal rupture may occur, and an acute picture then develops The classic acute syndrome is seen when intraperitoneal bleeding is prominent, and it may be associated with vascular collapse *The most important factors in early diagnosis of ruptured ectopic pregnancy are a high index of suspicion and the use of culdocentesis* The diagnosis must be considered in any woman in the reproductive period of life who complains of menstrual irregularity and pelvic pain of unknown origin *Conditions that must be considered in the differential diagnosis are* presented in Table 7-4

MANAGEMENT

1. If the patient is not in coma, she should be fully advised about the risks attendant upon her condition and about the various surgical procedures, including hysterectomy, which may be necessary Blood should be typed and crossmatched immediately, and an intravenous infusion begun If blood is not available, 5% dextrose in saline, serum albumin (25%), plasma, or plasma expanders may be used
2. *Culdocentesis is very helpful If the tap is positive, prompt surgical intervention* by laparotomy is indicated If the tap is negative but clinical findings are strongly suggestive, colpotomy or laparoscopy is advisable
3. If the patient is in shock, waste no preoperative time pouring in intravenous fluids Open the abdomen and stop the bleeding
4. Anesthesia choices may be limited They will depend on the urgency of the situation and availability of personnel and facilities In rare situations, local anesthesia or heavy narcosis may be the only available methods
5. The incision should be adequate A vertical midline incision gives the surgeon speedy access to the abdominal cavity and allows better exploration of the pelvis and abdomen
6. After the abdomen is opened, arrest the bleeding, compressing the bleeding site until the contralateral tube, both ovaries, and the uterus are inspected and

of abdominal pregnancy, unless the patient is opposed to laparotomy and is prepared to stay in the hospital throughout the course of her pregnancy As the pregnancy advances, extensive adhesions of the sac to the adjacent organs progressively develop, as do the vascular channels to the placenta The diag nosis of abdominal pregnancy may be made, before catastrophic bleeding develops, on the basis of several features

Physical Examination

Examination may reveal abnormalities such as the following
1. A mass beside the uterus, in which fetal parts can be easily felt
2. Marked displacement of the cervix anteriorly or posteriorly is noted on pelvic examination The cervix is often small and firm and may (rarely) be palpated posterior or anterior to the abdominal pregnancy mass, somewhat distinct from it
3. The presence of the fetus high in the abdomen and in a transverse position
4. Inability to palpate fetal parts through a patulous cervix
5. No contractions of the abdominal mass noted after the administration of an oxytocin infusion

X-Ray Examination

X-ray films may reveal one or more of the following characteristics
1. Eccentric position and transverse or oblique lie of the fetus
2. No evidence of a uterine wall around the fetus on a soft tissue film
3. The presence of fetal parts posterior to the anterior margin of the vertebral column of the mother on a lateral film
4. The demonstration of the uterus separately by hysterography

Sonar Diagnosis

Diagnosis by sonar is difficult and requires a thorough knowledge of the varia tions seen in intrauterine pregnancy As outlined by Kobayashi, diagnostic steps include the following
1. Careful identification of the uterus
2. Identification of the fetal head outside the uterus
3. Identification of an ectopic placenta
4. Identification of the fetal body

In Figure 7-19 these criteria appear to be met but it is actually a retro-verted incarcerated uterus that is distended anteriorly The cervix, displaced anteriorly, looks like a displaced uterus Criteria 2, 3 and 4 appear to be met

MANAGEMENT

Laparotomy is performed as soon as the diagnosis is made At least 2000 ml of blood should be available at operation A base line hemoglobin and hematocrit should be obtained before surgery

When possible, the fetus and amniotic sac are removed The umbilical cord should be ligated as close to the placenta as possible and the placenta should be left in position, the abdomen being closed without drainage If it is not pos sible to remove the sac following delivery of the fetus, the sac should be either

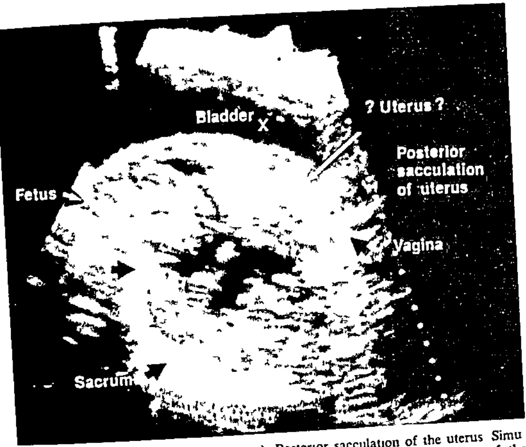


FIG 7 19 Sonogram (Saggital section) Posterior sacculation of the uterus Simulating extrauterine pregnancy The fetus is within the hollow of the sacrum, apparently extrinsic to an anteriorly displaced uterus, apparently compressing cervix and lower uterine segment Placenta not shown on this view

drained or marsupialized When severe and uncontrollable oozing occurs, the abdomen should be packed following the delivery of the fetus The pack should be brought out through the abdominal wall or, if possible, through the posterior cul de sac, to be removed under anesthesia after 48 hours

The main postoperative dangers are from recurrent hemorrhage, infection, and intestinal obstruction If anemia is present, the patient should be treated before discharge from the hospital

OVARIAN PREGNANCY

This is a rare form of ectopic pregnancy The primary type presumably results from intrafollicular fertilization Rupture occurs early, by the eighth or ninth week of amenorrhea, but occasionally an ovarian pregnancy may go on to near term and be detected only at the time of surgery for obstructed labor

Despite its rarity, the matter is of considerable interest because of the apparent increase in patients using intrauterine contraceptive devices Hemoperitoneum due to the rupture of an early ovarian pregnancy would not be clini-

cally distinguishable from that due to other forms of early ectopic pregnanc At surgery, if the tube is normal, and the bleeding is from the ovary, this p sibility must be kept in mind Usually, suture of the bleeding site is carried c Resection of the corpus luteum would be necessary to establish a tissue di nosis If the latter procedure is done, then there is no need for concern that resection would result in the abortion of an early, unrecognized intrautei pregnancy

POST-STERILIZATION ECTOPIC PREGNANCIES

Ectopic pregnancies may still occur in women who have had sterilizing p cedures Although the incidence is extremely low, the possibility must be c sidered in women in the reproductive years who develop acute pain in lower abdomen This applies whether sterilization was by hysterectomy or tu interruption, although its occurrence after hysterectomy is very rare inde Suprapubic transabdominal aspiration may be done after the bladder is empt to assist in the diagnosis of hemoperitoneum when the cul de-sac is not free

ISOIMMUNIZATION IN EARLY PREGNANCY HEMORRHAGE

The original concept of Rh disease envisioned the constant leakage of red c into the mother's circulation during the course of pregnancy Hence prim immunization could take place at any time It was shown eventually t sensitizing transplacental hemorrhages were mostly associated with delivery : placental separation Subsequently, it was shown that Rh immunoglobulin gi after delivery was very protective

Recent evidence suggests that, in early pregnancy, Rh incompatible fe to-maternal bleeding may occur, enough to initiate the sensitization phenome: even in a first pregnancy This may occur in cases of threatened abortion, topic pregnancy, and certainly when an elective abortion is done It has b proposed that in any of these situations, anti Rh_0(D) immunoglobi (Rhogam) be given to the Rh negative patient This is advisable when d nite likelihood of sensitization occurs, as in an elective abortion late in the i trimester

TROPHOBLASTIC DISEASE

Insofar as growth is concerned, chorionic tissue behaves in a pseudo-malign fashion even under normal circumstances It invades the uterine wall but oi narily stops at the decidua-myometrial junction Embolization occurs, but deposits do not behave in a malignant fashion The tissue itself may show biza histologic patterns, which vary through all stages of anaplasia and degr of proliferation They may be seen in any specimen of chorionic tissue

Gestational trophoblastic disease is a relatively frequent problem Benign a malignant forms have long been recognized The traditional clinical classifi tion is shown below It is based on morphological features and evidence invasiveness

1. Hydatidiform mole (benign with malignant potential)
2. Chorioadenoma destruens ("invasive mole")
3. Choriocarcinoma (malignant)

It is now generally recognized, however, that histopathologic features do *not* accurately predict malignant potential and this classification is obsolete. The difference between the relatively benign and very malignant forms are more accurately expressed in terms of biologic function, *i.e.*, production and excretion of human chorionic gonadotropin (HCG). Thus, variation from the excretion pattern for normal pregnancies such as is shown in Figures 7-2 and 7-3 suggest abnormal trophoblastic growth. Similarly, the reappearance of HCG in body fluids suggests renewed growth of trophoblast whether in utero or elsewhere.

HYDATIDIFORM MOLE

The classic mole is of major clinical importance because of the likelihood of hemorrhage and sepsis. The incidence varies greatly (United States, 1 in 2000 pregnancies; Mexico, 1 in 200 pregnancies; Taiwan, 1 in 125 pregnancies). Diagnosis is often not suspected until vesicles are passed or the abnormality is encountered in the treatment of what appears to be an inevitable abortion or even a premature labor.

Diagnostic Features

1. Intermittent-to-continuous brownish discharge punctuated by episodes of frank bleeding, this becoming more marked by the 12th week
2. Protracted nausea
3. Disproportionate growth of the uterus (50% of cases)
4. Absence of fetal movement or fetal heart tones even when the uterus is greater than 24 weeks in size (the possibility of twins must be kept in mind)
5. Absence of fetal parts and a boggy consistency of the uterus on pelvic examination
6. Bilateral ovarian enlargement due to lutein cyst formation in 25–60% of cases
7. Anemia may be present as a result of the recurrent external bleeding, the blood contained in the mass of chorionic tissue, hypervolemia, and dietary inadequacy
8. The appearance of pregnancy-induced hypertension, edema, and albuminuria before the 24th week of gestation, particularly if the uterus is above the umbilicus or large for dates
9. The passage of vesicles

Ancillary Studies

1. In normal pregnancy, during the first 100 days after the last menstrual period, urinary gonadotropin levels may be very high. After this time they seldom exceed 20,000 IU/24 hours (Figs. 7-2, 7-3). Persistently higher and rising titers after that time are strongly suggestive of abnormal trophoblastic proliferation.

2. By the 18th or 20th week of gestation fetal parts should be evident on an x-ray film of the abdomen If negative, the x-ray should be repeated in 2 weeks A honeycomb pattern to the uterine shadow is also suggestive of the diagnosis
3. A transabdominal amniography may be attempted in the manner of an amniocentesis If no fluid is obtained, this is suspicious, and 20 ml diatrizoate (Hypaque) is quickly injected Five to 10 min later, an anterior-posterior roentgenogram is made of the lower abdomen and pelvis A honeycomb pattern may be produced by contrast material interspersed between the vesicles
4. Sonar examination is a most effective way of establishing the diagnosis Transitional, partial, and complete moles can be identified relatively early (Fig 7-6, 7-7)

Management

Management is based on 1) the need to evacuate the mole, and 2) the need to follow the patient subsequently to detect malignant change in remaining trophoblastic tissue These steps apply in all cases, no matter what the histologic appearance of the tissue obtained because subsequent choriocarcinoma is variously reported to occur in 0 5–9 5% of all cases

Hemorrhage, sepsis, and possible uterine perforation pose the immediate clinical problems Hemoglobin, white count and differential base line values must be obtained Urinary output must be monitored Hemorrhagic shock must be treated if present on admission The specific approach to evacuation of the mole will depend upon when it is diagnosed, the condition of the cervix the amount of bleeding, the size of the uterus and whether the patient is in the process of aborting the mole

MOLAR EVACUATION

1. Small uterus (less than 12 weeks) Blood should be available Sufficient cervical dilatation to allow passage of a large bore vacuum suction curet can usually be safely accomplished Oxytocin infusion (Pitocin) 10 units in 1000 ml dextrose in water should be maintained throughout the procedure No uterine relaxing agents, such as halothane, should be used After the bulk of the tumor is removed, gentle but thorough sharp curettage should be done Oxytocin infusion should be continued until the uterus is well contracted bleeding has stopped and the patient has recovered from the procedure Ergonovine (Ergotrate) or methylergonovine (Methergine) 2 mg IM every 8 hrs for 3 doses may also be used
2. Large uterus The larger the mole, the more likely a patient is to have persistent trophoblastic disease (Morrow *et al* 1977) Severe hemorrhage may occur, either spontaneously or during evacuation If the patient is not already aborting, an oxytocin infusion may be used to initiate the process The solution used is 1000 ml of 5% dextrose in water with 10–20 units of oxytocin The drip rate is adjusted to effect uterine contractions If more than one infusion is necessary, the oxytocin may be increased by 10 units and the vehicle changed to saline or lactated Ringer's solution It must be remembered that excessive oxytocin (greater than 40 mU/min) and large volumes

of hypotonic solution may induce water intoxication with water retention, hyponatremia and convulsions

Once the abortive process is initiated, evacuation may be aided by finger, ring forceps, or vacuum suction curettage Nitrous oxide (40%) with oxygen (60%) analgesia may be adequate Bleeding will usually be heavy and transfusions may be necessary If the mole is evacuated without the use of suction curettage the uterus should be allowed to involute for several days, if possible, before a repeat sharp curettage is attempted Extreme care must be taken not to perforate the uterus, nor should the myometrium be damaged by too deep curettage, for fear of producing a subsequent Asherman's Syndrome

3 Hysterotomy Hysterotomy is rarely necessary today Suction curettage has become the recommended approach, and even a uterus over 30 weeks' size, containing a mole can be emptied within 2–3 minutes

In partial moles, a hysterotomy may be necessary because of the large size of the coexisting fetus and the excessive maternal hemorrhage

4 Hysterectomy In women over 40, or in those who have completed their childbearing, hysterectomy should be considered, in view of the relative frequency that choriocarcinoma develops in these groups

5 Ovarian enlargement Significant enlargement (greater than 8 cm) of the ovaries due to the presence of multiple theca lutein cysts occurs in 10–15% of patients These should generally be left alone, although it should be kept in mind that persistent trophoblastic disease is more common when these are present Occasionally torsion or hemorrhage into the ovary may create acute abdominal pain with evidence of peritoneal inflammation Then the involved adnexa must be removed Enlargement, especially in association with a large for-date uterus, may portend a greater risk of malignant sequelae

Following initial evacuation of a mole, approximately 90% of patients are cured Malignancy may develop in the other 10% It should be kept in mind that invasive trophoblastic disease can usually be traced back to hydatidiform mole (50%), abortion (25%), or term pregnancy (25%) It may even coexist with a normal pregnancy

CHORIOADENOMA DESTRUENS

Like hydatidiform mole, this is a type of trophoblastic disease

This locally invasive tumor may be responsible for continued uterine bleeding after a term pregnancy, abortion, or molar evacuation Perforation of the uterine wall with resulting intraperitoneal hemorrhage may occur Hysterectomy may have to be considered in management planning but as emphasized in the section on Treatment and Prognosis, new concepts are emerging with regard to trophoblastic disease

CHORIOCARCINOMA

This highly invasive chorionic tissue tumor is prone to metastasize Chromosomal studies indicate a tendency toward aneuploidy It is infrequent The behavioral pattern is unpredictable and manifestations may be bizarre Chemotherapy is highly effective even when extensive metastases are present (Fig 7-20)

FIG 7 20 A Initial chest radiograph of patient with metastatic choriocarcinoma B After 9 months of treatment gradual resolution of the lesions occurred C. No change is seen in left hilar or right lower lobe metastases over 4 additional months Only necrosis and fibrosis were noted in resected left hilar lesion (Libshitz HI Baber CE Hammond CB Obstet Gynecol 49 415 1977)

As emphasized in the section on Treatment and Prognosis later in this chapter, histologic appearance is now considered less important than biologic effect

PATIENT MONITORING IN TROPHOBLASTIC DISEASE

Because of the unpredictable nature of trophoblast it is advisable that all patients at risk of developing trophoblastic disease be closely followed, even though the initial disease has apparently been satisfactorily treated High risk patients include women who abort and in whom placental tissue shows evidence of molar change, women who continue to bleed irregularly or show uterine subinvolution after an abortion or normal delivery, and women who have an obvious hydatidiform mole

Current management methods include 1) early diagnosis and appropriate treatment of the specific type of clinical disorder 2) careful monitoring of patients at risk, 3) prompt and adequate chemotherapy when indicated It should be emphasized that chemotherapy administered prophylactically in benign trophoblastic disease prevents malignant sequelae, as Gones (1975) points out, it should not be used routinely for the following reasons

1. Most patients are cured by evacuation alone
2. Essentially all patients who develop malignant sequelae in the immediate postevacuation period can be cured by present techniques
3. Minimal drug toxicity is experienced by most patients, but severe reactions or even death may occur in an unusually sensitive patient
4. Cytotoxic agents are teratogenic, and so recessive mutagenic changes may occur

Careful clinical evaluation is important, but serial measurement of HCG is essential The standard urinary tests for pregnancy are only grossly quantitative They are of no help in detecting the relatively low levels of HCG production that may occur during the early phases of abnormal growth They should not be used as an index of regression Quantitative biossay and hemagglutination inhibition assays for HCG in serum (or urine) are more accurate They have been used successfully in many centers as the basis for diagnosis, for monitoring of treatment, and assessment of regression Currently, the use of radioimmunoassays on serum for the beta subunit of chorionic gonadotropin is recommended These are specific and very sensitive (to 2–10 mIU/ml) If not available locally, this assay may be obtained through one of the Regional Trophoblastic Disease centers

Any physician requiring information on the diagnosis or management of trophoblastic disease should contact the director at the nearest center The Regional Trophoblastic Disease Centers in the United States are located at the following institutions Duke University Medical Center, Durham North Carolina, Harvard Medical School, Boston, Massachusetts, Memorial Sloan-

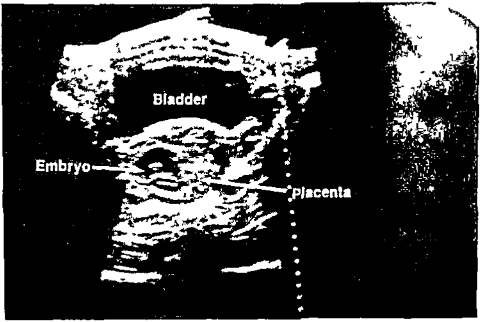


FIG 7 21 Sonogram (Transverse section) Intrauterine pregnancy one year after a hydatidiform mole

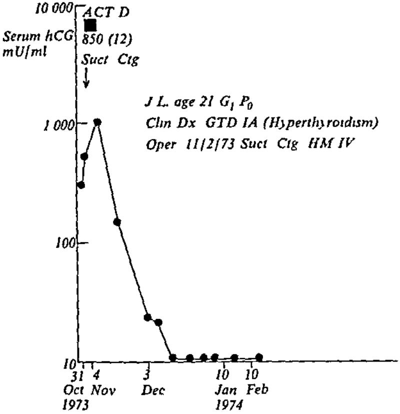


FIG 7-22 Goldstein DP Endocrine assay in chorionic tumors In Clinical Obstetrics and Gynecology Hagerstown Harper and Row, Vol 18, No 4, 1975, p 48

Kettering Cancer Center, New York, New York, Northwestern University Medical School, Chicago, Illinois, Roswell Park Memorial Institute, Buffalo, New York, University of Southern California, Los Angeles, California, and University of Texas, M D Anderson Hospital, Houston, Texas

A protocol is helpful in following these at-risk patients Depending on the nature of the clinical problem, modifications may be made, but the protocol should be followed in all at-risk situations

1. Patient must avoid pregnancy for 1 year Oral contraceptives may be used They will not interfere with chorionic gonadotropin testing If a new pregnancy is suspected, sonar examination is very helpful in clarifying the situation Recurrent moles are rare but they do occur (Fig 7-21)
2. Careful pelvic and general assessment should be made every 2 weeks to evaluate uterine and ovarian regression and to detect metastases
3. Determinations of HCG should be carried out on serum by radioimmunoassay for the beta subunit every 1–2 weeks depending on the trend As long as titers decline and reach normal within 60 days simple observation is satisfactory (Fig 7-22)
4. As long as titers are elevated, a chest x-ray every 4 weeks is advisable

5. When HCG is undetectable on two successive determinations 2 weeks apart, the disease may be considered to be in remission In the absence of complaints, clinical assessment and HCG determinations should be repeated at 2 to 3 month intervals for the subsequent year
6. If irregular uterine bleeding continues, with or without uterine subinvolution, a D&C should be performed regardless of the HCG titer
7. If during the 60 day observation period a 2-week plateau or a rise in HCG titers is detected, chemotherapy is advisable It should also be used if detectable titers reappear at a later date

TREATMENT AND PROGNOSIS IN TROPHOBLASTIC DISEASE

Successful treatment of malignant trophoblastic disease depends on 1) duration of the disease prior to chemotherapy, 2) magnitude of the immediate pretreatment HCG titer, 3) presence of metastases, especially cerebral or hepatic, and 4) proper administration of appropriate chemotherapeutic drugs

Trophoblastic disease has recently been classified in terms of these factors as shown below The main features of management and the prognosis for each type are summarized as follows

Group I—Nonmetastatic

Tumor confined to uterus

Titer and duration of disease of less importance than localization to uterus

All histologic diagnosis (*e g* , benign, premalignant or malignant mode), are treated in the same way

Incidence of choriocarcinoma, 10%

TREATMENT

1. Evacuation or hysterectomy
2. If elevated HCG titers persist, methotrexate (20 mg/day) or actinomycin D (10 μg/kg body weight for 5 days) with course repeated until HCG titers are negative on 3 successive determinations at 2-week intervals Courses of chemotherapy are given approximately every 7–15 days but the exact time depends upon recovery of the mucosa of the GI tract and the bone marrow

PROGNOSIS 100% remission

Group II—Metastatic (low risk)

Metastases, but not to liver or brain

Titer of HCG less than 100,000 IU/24 hours (or mIU/ml)

Duration of disease less than 4 months

All histological diagnoses treated in the same way

TREATMENT

1. Evacuation or hysterectomy
2. Single drug and sequential therapy

PROGNOSIS 80–90% remission

Group III—Metastatic (high risk)

Metastases, but not to liver or brain
Titer of HCG greater than 100,000 IU/24 hours (or mIU/ml)
Duration of disease over 4 months
All histologic diagnoses treated in the same way

TREATMENT
1. Evacuation or hysterectomy
2. Combination chemotherapy

PROGNOSIS 70–80% remission

Group IV—Metastatic (high risk)

Metastases, including liver and brain
All histologic diagnoses treated in the same way

TREATMENT
1. Evacuation or hysterectomy
2. Combination chemotherapy
3. Liver and/or brain irradiation

PROGNOSIS as high as 50% remission

LOCAL LESIONS CAUSING BLEEDING

Bleeding from the lower genital tract or from extragenital sites may simulate threatened abortion In the reproductive age group cancer is rare but may occur The causes are as follows

Lower Genital Tract Causes

1. Cervix
   - *Erosions*
   - Carcinoma
   - Trauma
2. Vagina
   - *Trauma*
   - Ulceration
   - Severe moniliasis
   - Varicosities
   - Carcinoma
3. Vulva
   - Trauma
   - Varicosities
   - Carcinoma

Extragenital Causes

1. Urinary tract
   - Renal lesions (hypernephroma)
   - Bladder lesions (cystitis, papilloma)
   - Urethral lesions (polyps, diverticula)
2. Bowel
   - Hemorrhoids
   - Rectal lesions (carcinoma, colitis polyps)

Ulceration of the Vagina

Severe vaginal ulceration may follow the use of potassium permanganate crystals to produce abortion The ulceration is caused by undissolved crystals in the douche solution Heavy bleeding from the local ulceration may occur Treatment is as follows

1. Vaginal irrigation with warm saline
2. Control of local hemorrhage by pack or hemostatic suture
3. Treatment of hemorrhagic shock by transfusion if necessary
4. Monitoring for intravascular hemolysis (see Chapter 2)
5. Complete blood count
6. Monitoring for hyperkalemia
7. Monitoring and treatment for local infection

CARCINOMA OF THE CERVIX

Carcinoma of the cervix occurs in about 1 in 2000 pregnancies Although rare, it may occasionally cause severe hemorrhage

Speculum examination will reveal the cause, and cauterization with acetone, in addition to a vaginal pack, will control the flow until the patient is admitted to the hospital

If bleeding continues, vaginal packing should be repeated If this fails and hemorrhage from the cervix continues, then hypogastric artery ligation may be required If the disease is considered to be international stage I or even II a radical hysterectomy should be considered if the patient's condition is satisfactory, and the surgeon skilled in the procedure If the fetus is over 20 weeks' size, it should be delivered by classic cesarean section before the other operative procedures are undertaken

Alternatively, the initial bleeding may be controlled by deep x ray therapy via a vaginal portal The vagina should be packed between daily treatments After 4000 rads tumor dose to the whole pelvis almost all patients abort spontaneously

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Chapter 8

Hemorrhage in Late Pregnancy

Denis Cavanagh, Ralph E. Woods

I am in blood
Stepp d in so far that should I wade no more
Returning were as tedious as go o er"
William Shakespeare (1564–1616)
Macbeth Act III sc 4

Hemorrhage in the last trimester should always be regarded seriously All antepartum patients should be instructed to report immediately to their physician any vaginal bleeding occurring at this time Most maternal deaths caused by placenta previa are due to the patient's (or her physician's) ignoring the "warning hemorrhages"

Spotting in the first trimester may be a prelude to hemorrhage in the third trimester No bleeding during pregnancy should be taken lightly If the patient is instructed to report any bleeding at any time, it allows the timely application of diagnostic procedures A number of relatively benign situations may be uncovered Sonar examination should probably be performed as part of this evaluation If carefully performed, it can, by the 16th week, indicate the location of the placenta and confirm fetal age In a number of cases, the placenta will be seen to encroach on the cervix If this is the case, subsequent examination is imperative because as the uterus grows, the edge of the placenta may lift away from the cervix

Even if third trimester bleeding is slight, patients should be transferred immediately to a hospital for full investigation No vaginal or rectal examination should be performed, and even a gentle speculum examination is better deferred until the patient is in a hospital with facilities for immediate transfusion, because hemorrhage can be torrential When bleeding of a severe nature occurs at home, immediate blood replacement is desirable If the facilities are available, transfusion should be started before or during transportation to the hospital Insofar as the mother is concerned, the most important conditions to be considered in late pregnancy hemorrhage are placenta previa, abruptio placentae, and uterine rupture (Table 8-1) However, any type of late-pregnancy hemorrhage represents a danger to the fetus and is associated with low birth weight (Table 8-2)

TABLE 8 1 Cause of Hemorrhage in Late Pregnancy in Patients Seen Over a 5-Year Period

Diagnosis	Patients No	Patients %
Rupture of marginal sinus (Abruption grade 0)	165	18 5
Abruptio placentae (Abruption grades 1 2 3)	121	13 6
Placenta previa	124	13 9
Rupture of the uterus	27	3 0
Cervicitis	11	1 2
Vasa previa	3	0 3
Circumvallate placenta	3	0 3
Vaginal ulceration	1	0 1
Vaginal varicosities	1	0 1
Cause undetermined	438	49 0
Total	894	

(From Cavanagh D and Talisman MR Prematurity and the Obstetrician New York Appleton Century Crofts 1969)

TABLE 8 2 Low Birth Weight and Late Pregnancy Hemorrhage

Late pregnancy hemorrhage	Birth weight <2500 g No	<2500 g %	>2500 g No	>2500 g %
Placenta previa	81	1 6	167	0 26
Abruptio placentae	143	2 8	174	0 27
Other	126	2 4	229	0 37
Total	350	6 8	570	0 9
Total babies born	5151		64 139	

(From Division of Health State of Missouri 1974)

Painless bleeding in a multiparous patient with an abnormal presentation and high presenting part suggests placenta previa On the other hand, abdominal pain with vaginal bleeding and a hypertonic and tender uterus in a preeclamptic primigravida is more suggestive of abruptio placentae In a patient who has had a previous cesarean section, the possibility of a ruptured uterus must be kept in mind Occasionally vaginal and cervical lesions cause heavy bleeding, as may also 'rupture of the marginal sinus' (marginal abruption) of the placenta The obstetrician should be aware that the patient may confuse hematuria or even rectal bleeding with vaginal bleeding, but these can usually be easily excluded If any doubt exists as to the origin of the bleeding, uterine origin must be assumed Generally, the specific diagnosis is

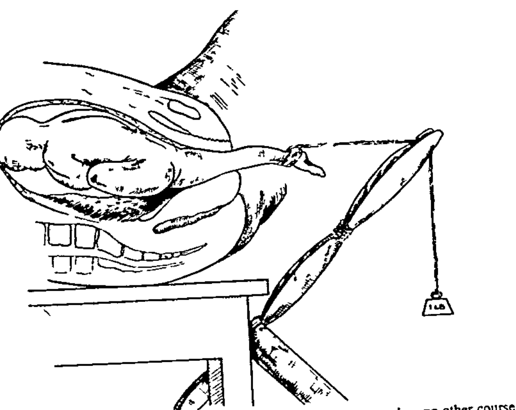

FIG 8 1 Placenta previa Control of bleeding by leg traction when no other course is available

not made until the hospital is reached and indeed, in about 50% of patients, no cause is found despite careful investigation

Very rarely the physician may be called to a home and find the patient exsanguinating because of severe vaginal bleeding In a dire emergency such as this, when the cervix is dilated only about 3–4 cm in the presence of placenta previa, it may be possible to effect adequate tamponade by applying leg traction to a fetus presenting in the breech position (Fig 8-1) When the vertex is presenting, the performance of external or even bipolar podalic version prior to pulling down the leg will of course be necessary (This technique must not be used as a substitute for hospitalization) Vaginal packing is of no value in controlling hemorrhage caused by placenta previa In these cases all efforts should be directed toward blood replacement and delivery

Ideally, only fully equipped hospitals with adequate resident staffs including anesthesiologists and pediatricians, should accept cases of antepartum hemorrhage On admission, the patient should be typed and matched for 1000 ml of blood, and base line hemoglobin and hematocrit estimations should be obtained When any significant degree of bleeding is present, no time should be wasted on admission paper work The patient must be taken immediately to the labor delivery unit The following treatment principles apply

1. Blood loss must be replaced with matched blood—not with plasma, saline, Ringer's lactate, or dextran These fluids should be used only when the patient is in shock and no matched blood is available at the time of admission,

or if a delay is anticipated When massive transfusion of blood is necessary, 44 6 mEq sodium bicarbonate should be given for every five units of blood to combat acidosis Also, two units of fresh frozen plasma should be given for each 10 units of blood to supply clotting factors which are frequently absent in banked blood (Fresh frozen plasma is unnecessary if fresh blood is available) Dextran is useful in the management of hypovolemia, but its administration may be associated with coagulation problems

2. Fluids should be given intravenously through an 18-gauge needle or an intravenous cannula so that blood in large quantities can be administered at any time There should be no hesitation in starting a second venoclysis or a cutdown when the physician has any doubt about keeping up with blood loss
3. Measurement of the central venous pressure or the pulmonary artery wedge pressure is very important in the management of hypovolemic shock These reflect the degree of cardiac competence (see Ch 3, Shock) For the measurement to be valid, catheters must be placed accurately, free flow ensured, and the same zero point of the manometer used at all times during monitoring
4. Any hospital accepting patients with antepartum hemorrhage should not only have facilities for blood transfusion but also blood available for immediate matching and use An emergency cesarean section cart should be kept fully equipped at all times to deal with cases of severe late pregnancy hemorrhage
5. An anesthesiologist should be available for consultation as to the type of anesthesia most suitable for patients who have experienced varying degrees of blood loss
6. Vaginal examination should be carried out only under double setup conditions—*i e* , in the operating room with preparations for performing immediate cesarean section, or rupture of the membranes, if necessary The patient should be "prepped" and draped, blood should be available, and an intravenous infusion of saline should be started through an 18-gauge needle or intravenous cannula Rectal examination should not be done, for it may stimulate heavy bleeding and is completely inadequate
7. A gentle speculum examination may be carried out when blood is available and an intravenous infusion has been started In a patient who has had an episode of severe vaginal bleeding and in whom the abdominal findings suggest a major degree of placenta previa, speculum examination should be postponed until "double set-up" examination is undertaken All patients who are allowed to return home should have a careful speculum examination before discharge This may reveal the presence of vaginal varicosities, ulceration of the vagina, cervical polyps or other lesions Papanicolaou smears and punch biopsies should be taken of all cervical lesions even at this stage in pregnancy The bleeding associated with cervical biopsies can usually *be controlled with a vaginal pack If no evidence of malignancy is found, the* cervical erosion may be treated with a silver nitrate stick
8. A careful abdominal examination should be performed Particular attention should be paid to the presentation, the height of the presenting part above the pelvic brim, and the presence or absence of increased uterine tone and tenderness The height of the fundus of the uterus should be measured repeatedly when abruptio placentae is suspected When the patient is in labor, the type of uterine contractions and the time between them should be noted If the indications are not those of a ruptured uterus or abruptio placentae, she should be regarded as having placenta previa until proved otherwise

As soon as the patient's general condition has improved sufficiently, a careful history and general physical examination should be performed. Particular attention should be paid to a history of hypertensive disease and to the presence of hypertension, edema, and proteinuria in the present pregnancy. The pulse and blood pressure should be carefully watched, and a urinalysis should be done. The parity of the patient, the amount and character of blood loss, and the presence or absence of abdominal pain are important factors to consider when making a clinical diagnosis. Whether the placenta is implanted anteriorly or posteriorly and the presence or absence of fetal heart tones will influence management in cases of marginal placenta previa.

The cardinal factor in the management of all patients with antepartum hemorrhage is the amount of bleeding, be it internal or external.

If the patient shows no further evidence of bleeding after arrival at the hospital, and the diagnosis is not obvious, her condition is managed as placenta previa. She is confined to bed even if bleeding has stopped. Placentography is utilized in an attempt to visualize the site of the placenta or to deduce its position. This may be done by ultrasonic, isotopic, or roentgenographic methods. When available, sonar is the preferred method for establishing placental location in relation to the cervix. When the diagnosis remains in doubt, no patient should be discharged from the hospital without a speculum and double setup examination.

PLACENTA PREVIA

Placenta previa is said to be present when all or part of the placenta lies in the lower uterine segment. The outcomes for the mother and child depend largely upon the degree of placenta previa when the cervix is fully dilated.

CLASSIFICATION

No ideal classification has yet been introduced. In relationship to the ultimate prognosis, four types are best described (Fig. 8-2).

Type I. This is merely a low implantation of the placenta with the inferior border extending into the lower uterine segment but not reaching the margin of the internal os. In this case, the placental edge can be palpated by the tip of the examining finger introduced through the cervical canal at double setup examination. For example, with the cervix 1–2 cm dilated, the lower edge of the placenta would lie within 4 cm of the internal os.

Type II. The placental edge reaches the margin of the internal os when the cervix is fully dilated.

Type III. The placenta partially covers the internal os when the cervix is fully dilated.

Type IV. The placenta completely covers the internal os when the cervix is fully dilated.

These classifications involve a projection of the situation as it would be at full dilatation of the cervix, on the basis of the pelvic findings at the initial vaginal examination. This approach provides a greater margin of safety for the woman and her fetus. Attention should also be paid to the location of the placenta—anterior or posterior—because a patient with a placenta implanted posteriorly is less suitable for vaginal delivery.

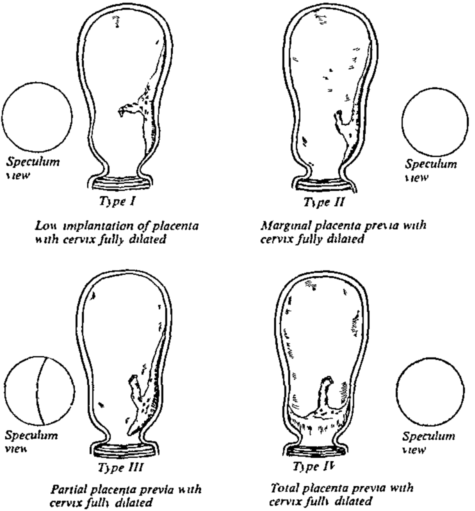

FIG 8 2 Placenta previa Classification based upon the anticipated situation of the placenta at full dilatation of the cervix.

The standard classification into marginal partial, and total placenta previa on the other hand, is based directly upon the findings at the initial vaginal examination and without regard to the stage of cervical dilatation The danger of using the latter classification lies in the possibility of minimizing the degree of placenta previa For example, a placenta that is palpable at the margin of the os at 2 cm cervical dilatation will probably be a partial placenta previa at full dilatation

On vaginal examination, it is sometimes difficult to differentiate blood clot from placenta It is emphasized that irrespective of the classification used the obstetrician must try to visualize the situation that will exist at full dilatation of the cervix, on the basis of observations at digital examination of the vagina during double setup examination

ETIOLOGY

The cause is unknown, but faulty implantation, endometritis following a previous pregnancy, uterine scars, and repeated pregnancies with short intervals between have been implicated

INCIDENCE

Placenta previa complicates about one in every 200 pregnancies Eighty percent of the patients are multiparas, and the occurrence rate rises with parity, so that it complicates about 5% of pregnancies in women who have had five previous pregnancies However, this appears to be an effect of age, as the condition is three times as common in women over 35 as in women under 25, regardless of parity Placenta previa tends to recur

DIAGNOSIS

A history of painless vaginal bleeding with the passage of bright red blood during the last trimester of pregnancy should immediately suggest placenta previa Frequently, the bleeding occurs while the patient is in bed, and the more serious the degree of placenta previa, the earlier in pregnancy will bleeding be likely to occur It is extremely unusual for a patient to have a sudden torrential hemorrhage without preceding "warning' hemorrhages It is almost unknown for a woman to die after the first episode of bleeding unless it has been precipitated by an ill advised vaginal or rectal examination Coitus may also precipitate heavy bleeding

The most significant advance in the management of placenta previa occurred in 1927 In that year, Bill pointed out that the complication carried a maternal mortality of 10%, and he advocated the liberal use of blood transfusion and the more frequent performance of cesarean section

About 90% of women with placenta previa will have at least one significant bout of late pregnancy hemorrhage, and 10–25% will develop shock Patients with the more extensive types of placenta previa tend to bleed earlier and more heavily, 50% of patients with type IV (central) placenta previa have the initial bleeding prior to 30 weeks

Abdominal Examination

Uterine tone appears normal with no tenderness The presenting part will usually be found floating high above the symphysis pubis, and it cannot be pushed down into the pelvis The presence of a breech or shoulder presentation is not uncommon

Ancillary Methods

In almost all cases a double setup vaginal examination is necessary to establish the diagnosis and decide upon the method of management If bleeding is not marked and the baby is very small, ancillary methods may be used to make a presumptive diagnosis

ROENTGENOGRAMS X ray films are of value in determining whether placenta previa is present after the 34th week of pregnancy They are somewhat unreliable however in the first half of the third trimester when the clinician is most in need of diagnostic aid They should not be relied upon in decid ng the degree of previa present

Direct Placentography Soft tissue placentography is probably the most popular method The placental outl ne can be demonstrated in most cases (Fig 8 3) unless it is obliterated by the bony pelvis The detection of a definite placental shadow in the upper uterine segment will exclude placenta previa and save much worry for both the pat ent and physician It is emphasized that success with this method depends entirely upon good roentgenographic techn que and interpretation

FIG 8 3 D rect (soft t ssue) placentography w th an anteroposterior film show ng placenta prev a (Cavanagh D Tal sman MR Prematurity and the Obstetr cian Englewood Cl ffs Prent ce Hall 1969)

Indirect Placentography This method is based upon deduction of the placental position from displacement of the fetal head If the presenting part lies centrally in the anteroposterior view, and within 2 cm of both the sacral promontory and the symphysis pubis on the lateral view, placenta previa can be excluded If in the erect lateral film the presenting part is found to be over 2 cm from the sacral promontory, the patient is placed in the semierect position and another lateral film is taken If, despite the patient's change in position, the gap between the presenting part and the promontory is still greater than 2 cm, the presence of a posterior placenta previa overlapping the sacrum can be deduced (Fig 8-4 to 8 6) In general, this is the most satisfactory radiographic method, and it has the advantage of being relatively independent of the quality of radiography The main disadvantage is that the taking of these films increases the radiation hazard

FIG 8-4 The basis of *indirect placentography* is the *cephalopelvic relationship in* the presence of a posteriorly implanted placenta previa (Cavanagh D Talisman MR Prematurity and the Obstetrician Englewood Cliffs Prentice Hall 1969)

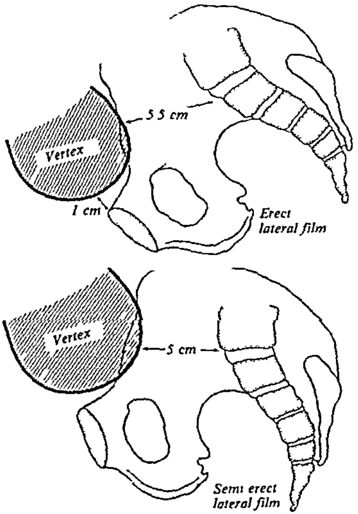

FIG 8 5 Indirect placentography with an anteroposterior film taken with rad o-paque dye in the bladder and a r in the rectum demonstrating displace ment of the vertex by placenta previa (Cavanagh D Tal sman MR Prematurity and the Obstetric an Englewood Cliffs Prent ce Hall 1969)

Cystography This is now rarely used The bladder may be filled with 12% sodium iodide solution but distention with about 300 ml of air introduced through a catheter is equally satisfactory A lateral roentgenogram is taken to determine the distance between the bladder shadow and the fetal skull When this exceeds 1 5 cm the presence of placenta previa with implantation on the anterior wall of the uterus should be suspected

Amniography The instillat on of radiopaque material into the amniotic sac may be used to demonstrate the presence of the placenta as a filling defect This method is now rarely used

Placental Arteriography Although this is a highly accurate method of placental localization it is rarely used because of the h gh dose of radiation to the fetus and the hazards to the mother

FIG 8 6 Indirect placentography, with lateral roentgenogram taken with radiopaque material in the bladder and air in the rectum demonstrating displacement of the vertex by placenta previa (Cavanagh D Talisman MR Prematurity and the Obstetrician Englewood Cliffs, Prentice Hall, 1969)

THERMOGRAPHY This method is based on the presence of increased skin temperature over a vascular area The method is safe, but its reliability is unproved

ISOTOPIC LOCALIZATION This technique subjects the mother and the baby to less irradiation than x-ray placentography Radioiodinated serum albumin, chromium-51 technetium-99-albuminate, and radioactive indium have all been used for this purpose The last two are the most desirable isotopes for use, but all methods are based on the pooling of the isotope in the intervillous space The uterus is marked (Fig 8-7), the isotope is injected intravenously, and the scan is made, as in Figure 8-8, in which a technetium scan demonstrates placenta

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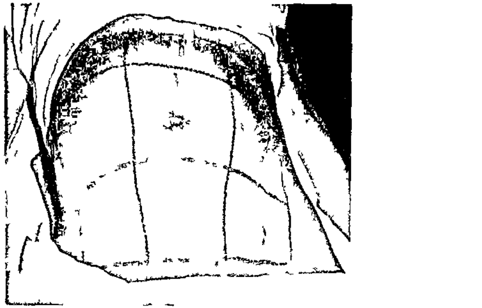
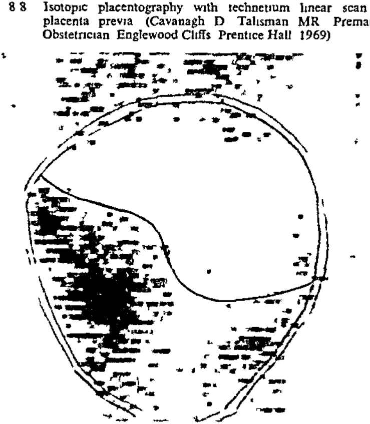


FIG 8 7 Uterus is outlined and overlying skin is marked off using a skin pencil or felt tipped pen. (Cavanagh D Gilson A Powe E South Med J 54 1340 1961)

FIG 8 8 Isotopic placentography with technetium linear scan demonstrating placenta previa (Cavanagh D Talisman MR Prematurity and the Obstetrician Englewood Cliffs Prentice Hall 1969)



ULTRASONIC PLACENTOGRAPHY This technique has superseded all others where the equipment and personnel skilled in its use are available The technique is accurate, does not involve any radiation hazards, and no fetal damage has been demonstrated to date Thus repeated examinations can be performed An accuracy rate as high as 97% in localizing the placenta has been reported Placental migration has been described, and King (1973) is of the opinion that demonstration of placenta previa before late third trimester calls for a reexamination at term

Repeated sonograms also provide evidence concerning fetal growth on the basis of serial measurements of the biparietal diameter of the fetal head Figures 8 9 and 8 10 show ultrasonograms in two patients with placenta previa

A vaginal examination must be carried out at some time on every patient with placenta previa to make the final diagnosis and to determine the degree of previa and the condition of the cervix The decision as to the method of delivery will depend largely upon these findings In all cases this vaginal examination must be performed in the operating room It should be preceded by a careful speculum examination to exclude a vaginal or cervical lesion An isotonic saline or Ringer's lactate intravenous infusion should have been commenced using an 18 gauge needle or intravenous cannula and 1000 ml of matched blood must be immediately available The patient should be prepared, draped, and ready for immediate cesarean section in case torrential hemorrhage is precipitated by the examining finger

MANAGEMENT

Since 1945 when Johnson and MacAfee cited success with the expectant (conservative) management of placenta previa, this approach has become widely adopted The method was introduced for the sole purpose of improving perinatal survival and requires that the pregnant woman be restricted to almost complete bed rest from the first bleeding episode and presumptive diagnosis of placenta previa until the fetus reaches the age of 36 weeks and an estimated weight of 2500 g

Recently the concept has come under strong attack In the face of mounting evidence, it is difficult to justify it except in certain circumstances, as when a good neonatal intensive care unit is not available However, a controlled study has not yet been carried out Moreover, first class intensive care facilities for newborns are not universally available In view of this, the conservative approach will continue to be used The procedure is as follows

When the patient arrives at the hospital and placenta previa is suspected, the primary factor influencing treatment is the amount of bleeding

If Bleeding Stops

When the patient is less than 36 weeks pregnant, the baby's weight is estimated to be less than 2500 g (5 5 lb), and the patient is not in labor, expectant treatment is a reasonable course, and pelvic examination must be avoided

1. The patient is confined to bed for at least 72 hours During this time, examinations should be carried out in an effort to establish the diagnosis
2. Blood transfusion should be given as necessary to keep the patient's hemo-

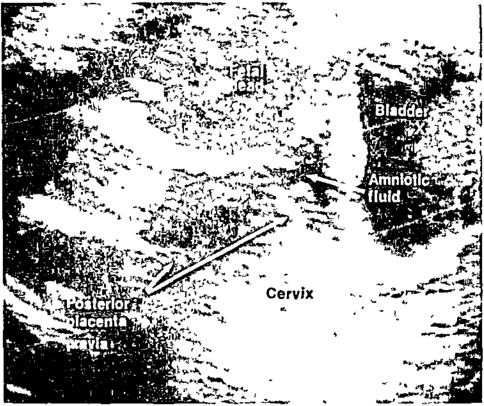
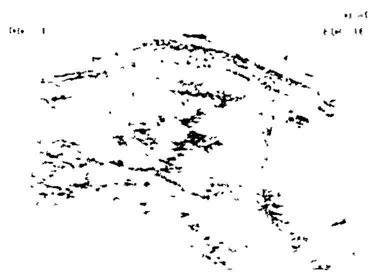


FIG. 8-9. Sonogram showing posterior placenta previa covering most of internal cervical os.

FIG. 8-10. Sonogram showing complete placenta previa symptomatic at 24 weeks from last menstrual period.



globin at a minimum of 10 g/100 ml At least 1000 ml of matched blood should be available at all times until 24 hours after delivery

3. The patient may be allowed to ambulate after the third day, even if the placentogram suggests placenta previa However, transfusion and confinement to bed may be necessary if further bleeding occurs
4. Amniocentesis should be performed and an estimation of fetal maturity obtained
5. If a diagnosis of placenta previa is made by an accurate ancillary method, the patient should be advised to remain in the hospital at almost complete bed rest. When this is economically impractical or if the patient insists on leaving the hospital for any reason she should be told of the danger of sudden severe hemorrhage Her relatives should be warned that she must have a companion with her at all times and that she must return to the hospital at the slightest sign of bleeding She should stay in bed as much as possible Coitus and douching are strictly forbidden Matched blood should be kept available for these patients even after they have left the hospital

   Unless the fetus is very immature if it seems likely that the patient will leave the hospital, then for the safety of the woman and child it is advisable to perform a double setup examination and proceed to delivery by the most expeditious route
6. If the fetus is estimated to be at least 36 weeks old and 2500 g in weight and an x ray or sonographic diagnosis of placenta previa has been made, a vaginal examination with double setup should be done The vaginal examination should be preceded by a careful speculum examination
7. If at the time of sterile vaginal examination the placenta is found to be covering the internal os completely or partially a major degree of placenta previa (type III or type IV) is present and cesarean section should be carried out at once. When the placental edge is palpable within 5 cm of the internal os before the onset of labor type I or II placenta previa is usually present In these cases if the fetus is viable, cesarean section is the treatment of choice In a patient with type I or type II placenta previa and a small or dead fetus, vaginal delivery may be safely effected, provided bleeding is not excessive, and the placenta is not implanted on the posterior wall of the uterus The increased hazard of cervical and uterine lacerations following the vaginal delivery of a patient with placenta previa must not be forgotten

Again it is emphasized that before a vaginal examination is undertaken on a patient suspected of having placenta previa all preparations should be made for an emergency cesarean section, with blood available and an intravenous saline infusion started (double setup)

If on admission, the patient's pregnancy has advanced beyond 36 weeks and fetal weight is estimated to be 2500 g or over, there is no need for expectant management, and the patient should be examined vaginally in the operating room as soon as possible after admission even if vaginal bleeding has stopped In this situation cesarean section is again the treatment of choice, as it is for all but the mildest types of placenta previa When a patient is in good labor with a minor degree of placenta previa, vaginal delivery may be allowed, provided the placenta is on the anterior or lateral uterine wall and bleeding is not excessive

If vaginal delivery is to be allowed the patient should be given oxygen by mask until the cord is cut This will help to protect the fetus from hypoxia The membranes may be ruptured artificially to initiate labor or to accelerate it If labor is poor, an oxytocin infusion may be given to improve it, but it should be

used only when contraindications—e g , transverse lie of the fetus—have been excluded Labor should be terminated by low forceps or assisted breech delivery If fetal distress develops, midforceps delivery or assisted breech extraction is justifiable, but the uterus must be explored after delivery to exclude laceration

If Bleeding Continues or Recurs

If recurrent or continuing bleeding is profuse, there is no room for expectant treatment, irrespective of the gestational age or state of the fetus

Double setup vaginal examination should generally be carried out in the operating room with preparations made for immediate cesarean section If bleeding is very profuse, double setup examination may have to be omitted Further management will depend upon the degree of placenta previa and the condition of the cervix

It must again be emphasized that at the time of double setup examination, a very careful speculum examination should precede introduction of the finger into the cervical canal If this precaution is taken, the obstetrician may be saved the embarrassment of performing a cesarean section on a woman who is bleed ing from cervicitis, cervical carcinoma, permanganate ulceration, or vaginal varicosities

At the time of cesarean section, the choice of operation will depend upon conditions present When the baby is small, it is probably undesirable to perform a low transverse cesarean section in the presence of an anterior placenta previa, for this entails cutting through placental tissue or separation of the placenta before delivery In this situation the baby loses blood, and a loss of even 30 ml in a premature infant may result in shock, and the child will require transfusion shortly after birth If a transverse lie is present an attempt at conversion to a breech or vertex presentation should be made while the abdomen is open If this fails, a longitudinal lower uterine incision or a classic operation should be performed Generally, a lower uterine segment operation can be performed safely in the presence of placenta previa This operation provides good exposure of the site of placental attachment, so that persistent bleeding can be controlled by sutures or direct pressure Very rarely is total hysterectomy or hypogastric artery ligation necessary

It is highly desirable to have a pediatrician available to take care of the baby at delivery, whether it has been delivered vaginally or abdominally

PROGNOSIS

With adequate antepartum care, the maternal mortality associated with placenta previa has been reduced to less than 1%, but maternal morbidity occurs in approximately 20%

The fetal perinatal mortality remains approximately 20% as a result of prematurity, intrauterine hypoxia, and developmental anomalies The presence of intrauterine death or skeletal abnormalities can usually be detected by ultrasonograms or by careful scrutiny of the films taken for localization of the placenta The presence of a dead or abnormal child will of course influence the choice of method for delivery

Despite efforts to improve perinatal survival in placenta previa, the mortality remains high and has not changed appreciably over the past 20 years This is one of the reasons that, with the availability of better intensive care neonatal units,

consideration is being given to early delivery rather than expectant treatment Although there is no relationship between fetal death and the number of hemorrhagic episodes, fetal death is sometimes related to the amount of blood loss In most cases the original bleeding episode occurs at a time when the fetus has a good chance for survival Thus, the general feeling now is that immediate cesarean section should be performed in a patient whose baby's gestational age is 35 weeks or more As Bishop (1974) has stated, "It must be recognized that except under the most ideal circumstances—circumstances which are difficult to achieve—it is difficult to document an improvement in perinatal mortality rate resulting from conservative therapy"

Furthermore, in a study of the impact of placenta previa on survivorship of offspring up to 4 years of age, Schlesinger and associates studied nearly 15,000 cases of placenta previa and 837,000 controlled births in upstate New York Placenta previa infants were found to be at considerably greater risk of dying during the perinatal period than other infants of the same birth weight and gestational age After early infancy, placenta previa infants were found to be at no greater risk of dying than other infants in the same birth weight and gestational age categories, so the occurrence of placenta previa had no impact on survival after 1 month of age This study suggests that coexistence with placenta previa is dangerous for the baby and that the danger is immediate

ABRUPTIO PLACENTAE

Abruptio placentae occurs when a normally situated placenta undergoes separation from the uterine implantation site, with resultant retroplacental bleeding after the 20th week of pregnancy and before the birth of the fetus The condition is also known as accidental hemorrhage and premature separation of the placenta

ETIOLOGY

Abruptio placentae appears to be associated with hypertension of any origin and increases with parity but not necessarily with maternal age Other factors that have been indicted in the etiology are external trauma, sudden uterine decompression, short umbilical cord, and uterine leiomyomas and anomalies Compression of the inferior vena cava and folic acid deficiency have also been blamed but there now appears to be little support for their inclusion as causative factors

INCIDENCE

The incidence of abruptio placentae is approximately 1 in 250 pregnancies The condition tends to recur in subsequent pregnancies Approximately 50% of abruptions occur before the 36th week of pregnancy

IMPORTANCE

The maternal mortality in abruptio placentae is approximately 2% if marginal abruption is excluded In severe cases associated with fetal death, maternal mortality has been reported as approximately 10% Perinatal mortality occurs

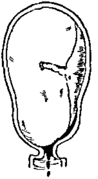
in over 50% of cases of abruptio placentae However, although it might be expected that increased morbidity would occur in surviving infants, there is no evidence that this is so

CLASSIFICATION

Three types of bleeding are usually described, and the clinical picture depends largely upon the type of bleeding (Fig 8-11)

In the revealed type of abruption, the amount of external blood loss is con sistent with the patient's general condition Hypertonicity of the uterus and uterine tenderness are not prominent features In the concealed type, no vaginal bleeding is seen, but the patient's general condition is much more seri ous than in the revealed variety, the uterus is tender its tone is markedly increased, and usually no fetal heart tones are heard Fortunately, the mixed type of abruption is much more common than the concealed variety, for the

FIG 8 11 Abruptio placentae Revealed and concealed bleeding



Revealed hemorrhage

Concealed & revealed hemorrhage

Concealed hemorrhage

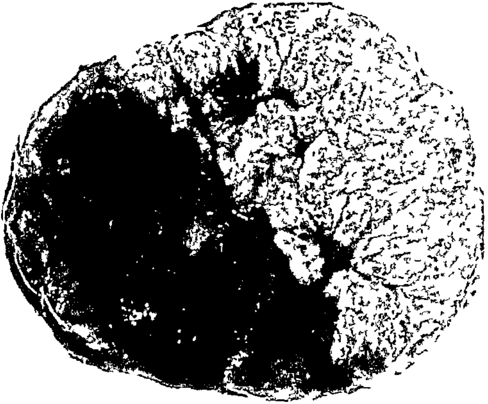
Premature separation of placenta with concealed hemorrhage

patient's complaints of vaginal bleeding and abdominal pain immediately suggest the possibility of abruptio placentae

Placental abruption is classified on the basis of severity from grade 0 to 3 grade 0 diagnosed only after delivery, when a small retroplacental clot is discovered The patients are usually asymptomatic, and those with so-called "rupture of the marginal sinus" are included in this category Grades 1 through 3 are distinguished on the basis of the amount of bleeding, the presence or absence of maternal shock, and the presence or absence of fetal death Thus, a patient with abruptio placentae grade 2 is a patient in shock but in whom fetal heart tones are still present The worst cases are associated with death of the fetus

Figure 8-12 shows clot adhering to a placenta, an indication that about 50% of this placenta was separated In addition to the fact that placental separation occurs, respiratory exchange over the attached portion of the placenta is also reduced as a result of vasospasm and disruption of the villi

FIG 8 12 *Abruptio placentae At left, separated portion shows typical appearance with adherent clot* (Ferguson JH Hatton RL Am J Obstet Gynecol 78 947, Copyright 1959, American Medical Association)



in the attached area It is not surprising that the fetus attached to this placenta died *in utero* (grade 3 abruption)

DIAGNOSIS

The terms revealed, concealed, and mixed refer to the location of bleeding rather than the degree of bleeding Here we are concerned mainly with the "mixed type of placental abruption in which the diagnosis is apparent with the severity classified as mild (grade 1), moderate (grade 2) or severe (grade 3) Marginal abruption (grade 0) presents a different picture and will be discussed as ' rupture of the marginal sinus " The concealed type eventually shows a revealed element when the blood separates the membranes from the uterine wall down to the internal os, and the ball-valve action of the fetal presenting part fails

History

Certain factors predispose to the development of placental abruption Toxemia of pregnancy, hypertension from any cause (particularly from cardiorenal disease) and polyhydramnios are the most common of these

Typically, the patient is engaged in some activity when abdominal pain develops This is followed by the onset of dark vaginal bleeding often with clots

Physical Examination

In severe cases the patient is often in a state of shock when first seen, and the shock is frequently out of proportion to the amount of external bleeding Localized uterine tenderness may become generalized as further retroplacental bleeding occurs When the patient is in labor, the uterus remains hypertonic or relaxes poorly between contractions In the presence of severe abruption the fetal heart tones are usually absent In the absence of abdominal pain and uterine tenderness, the vaginal bleeding is almost always due to some other cause such as ' rupture of the marginal sinus" or placenta previa It is essential to differentiate abruptio placentae from placenta previa for purposes of management (Table 8-3)

MANAGEMENT

Any patient in whom a diagnosis of abruptio placentae is made should be admitted directly to the labor-delivery unit Her history should be taken, and a careful but rapid assessment of her condition should be made Particular attention must be paid to pulse and blood pressure, and to the amount and type of vaginal bleeding A central venous catheter should be inserted into the antecubital vein and the central venous pressure should be carefully recorded since this is a better guide to blood replacement than the pulse, blood pressure or urinary output in this condition The patient's abdomen should be carefully examined, particularly with regard to uterine tone and tenderness, and the height of the uterine fundus should be measured (in severe placental abruption with retention of blood in the uterus, gradual enlargement in the

TABLE 8-3. Differentiation Between Abruptio Placentae and Placenta Previa

	Abruptio placentae (grades 1–3)	Placenta previa
History	Frequent association of toxemia of pregnancy or hypertension from any cause	No association with toxemia
	A single attack of vaginal bleeding, which usually continues until delivery	Repeated "warning" hemorrhages, often occurring over a period of weeks
	Abdominal pain	Usually no abdominal pain
Abdominal examination	Local uterine tenderness, hypertonic "woody" uterus in a concealed abruption	Normal uterine tone and usually no tenderness
	Patient usually in labor	Patient rarely in labor
	Presenting part often engaged	Presenting part above brim, malpresentations frequently found
	Fetal parts may be difficult to palpate	Fetal parts usually palpable
	Fetal heart tones often absent	Fetal heart tones usually present
Ancillary aids	Placenta demonstrated in upper uterine segment by ultrasonic, radiographic, or isotope studies	Placenta demonstrated in lower uterine segment by ultrasonic, radiographic, or isotope studies
Vaginal examination	Double setup reveals no placenta within 5 cm of internal os	Double setup reveals placenta implanted in lower uterine segment
Management	Admit all patients to hospital, no place for expectant treatment when this diagnosis is made	Admit all patients to hospital, if bleeding stops and fetus is less than 36 wk old, expectant treatment may be indicated

(Revised from Cavanagh D Obstetrical Emergencies 1st ed Springfield, Thomas, 1961)

uterus may occur) The fetal heart sounds must be carefully noted and should be externally monitored if equipment is available The quality of fetal heart tones may influence the mode of delivery If facilities are available uterine contractions should be monitored continuously Evidence of fetal distress and poor quality of uterine contractions are ind cations for cesarean section

It is obviously better to have a live premature baby than a fetal death *in utero* Thus once a definite diagnosis of abruptio placentae has been made for the sake of the baby as well as for the sake of the mother there is no place for expectant treatment Hemorrhagic shock must be treated immediately and the uterus evacuated as soon as possible Every effort must be made to recognize complications and to treat them adequately

Transfusion

The most important initial factor in therapy is early and adequate blood trans fusion A base line hemoglobin and hematocrit should be obtained and at least 2000 ml of blood should be crossmatched for use Oxygen should be g ven by catheter or mask for this will benefit both the mother and the fetus

Almost everyone knows about central venous pressure monitoring in septic shock but actually the central venous pressure is not as reliable a guide to fluid replacement in endotoxic shock as it is to fluid replacement in abruptio placentae O Driscoll *et al* (1966) at the National Maternity Hospital in Dublin were responsible for the introduction of central venous pressure monitoring in abruptio placentae and this was a very significant contribution to the management From the analysis of patients in their series it was the conclusion of O Driscoll *et al* that central venous pressure monitoring was a much more reliable guide than arterial pressure for the early detection of blood loss particularly in patients with concealed hemorrhage Most patients w th abruptio placentae are undertransfused The women in their series of 13 patients required an average of 7 units of blood with a range of 3 12 units This important fact must be kept in mind when assessing blood replacement needs in the patient with abruptio placentae

Coagulation Problems

A typical patient with abruptio placentae (Fig 8 13) had essential hypertension and superimposed toxemia You will notice that there are several tubes of blood on the wall behind the patient For a long time it has been taught that one of the compl cations of abruptio placentae is hypofibrinogenemia Ac tually this is somewhat inaccurate because the patient may actually have fibrinogen levels that are quantitatively in the normal range even in the presence of a severe coagulation problem The coagulation and fibrinolytic mechanisms and the diagnosis and management of the intravascular coagula tion/fibrinolysis syndrome (ICF or DIC) have already been detailed (see Ch 2 Clotting Disorders in Pregnancy)

In abruptio placentae thromboplastic material escapes into the maternal circulat on causing intravascular coagulation which itself leads to a tendency to further intravascular coagulation because the breakdown of fibrinogen and fibrin l berates fibrin split products This produces a hemorrhagic diathesis the presence of fibrin split products can be combated by giving heparin In

FIG 8 13 Patient with abruptio placentae Patient had toxemia of pregnancy superimposed on essential hypertension Note tubes of blood on the wall behind the patient for clot observation purposes (Ferguson JH Hatton RL Am J Obstet Gynecol 78 947 Copyright 1959, American Medical Association)

relatively chronic situations, such as the intrauterine dead fetus syndrome, this is good management for the defibrination syndrome, but heparin is rarely indicated in abruptio placentae

As already mentioned, the coagulopathy associated with abruptio placentae is often incorrectly described as a primary hypofibrinogenemia However, hypofibrinogenemia is usually present secondarily, and it is generally agreed that fibrinogen should be replaced if the level is under 100 mg/100 ml, and if blood loss is continuing or an operative procedure is contemplated Cryoprecipitate should be used in preference to fibrinogen because of the danger of serum hepatitis from fibrinogen

The mechanism of hypofibrinogenemia in pregnancy is threefold (Fig 8-14) 1) decreased synthesis of fibrinogen in the liver associated with reduced perfusion, 2) consumption of fibrinogen by intravascular coagulation, and 3) degradation of fibrinogen by fibrinolysin which may be primary or secondary (in obstetric cases it should be considered to be secondary)

These theoretic considerations do have practical applications If we compare fibrinogen with the fibrinogen first derivative, the substances are similar and are not differentiated in the quantitative test The important clinical application is that the clotting time is normal in the presence of fibrinogen but is prolonged in the presence of the first derivative Even if clotting does occur, the clot is unstable Electron microscopy reveals that in an abnormal clot, the defective polymer causes a "frayed rope" appearance Thus, a patient with a normal

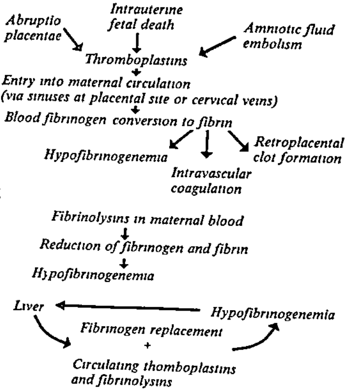


FIG 8 14 Possible mechanisms leading to hypofibrinogenemia A The thromboplastic mechanism B The fibrinolytic mechanism C. The hepatic depletion mechanism Normally the liver can replace the total blood fibrinogen within 24 hr If processes A and B continue, however, the liver cannot meet the demand (From I Kantor s discussion of the paper by Barno *et al* in Am J Obst Gynecol 77 1199, 1959 Copyright C V Mosby Company)

clotting time may have an abnormal clot Also, a patient with a fibrinogen level of over 150 mg/100 ml may still have a serious coagulation defect Thus, while it is true that an abnormality of blood coagulation may be indicated by hypofibrinogenemia, it must be appreciated that a severe defibrination syndrome may be present with even a fibrinogen level above 200 mg/100 ml

The presence of the intravascular coagulation/fibrinolysis syndrome (ICF)—*i e* disseminated intravascular coagulation (DIC) is indicated by a fall in platelets, a fall in prothrombin, a fall in factors V, VIII and XIII and the presence of fibrin split products in the circulating blood It has now been established that a coagulation defect is present in approximately 60% of patients with severe abruptio placentae This type of coagulation defect is also seen with amniotic fluid embolism (infusion), intrauterine dead fetus syndrome, eclamptogenic toxemia or septic shock

ICF (DIC) There continues to be a great deal of disagreement as to what abruptio placentae does to the normally elevated levels of fibrinogen in preg-

nancy At one time it was thought that the fibrinogen was consumed within the retroplacental clot However, recent studies have shown that there is no more fibrin within the clot that would have been found in the same amount of circulating whole blood A once popular theory is that there are elevated levels of circulating fibrinolysins that destroy fibrinogen but the weight of evidence now suggests that the fibrinolysis is secondary Also, when plasma from a patient who has ICF (DIC) syndrome is examined, it is extremely rare to find an increase in clot lysis The best explanation is that thromboplastins from amniotic fluid, or from the degenerating decidua, enter the maternal circulation and cause intravascular coagulation This view is supported by the fact that there is a decrease in multiple clotting factors and not merely in fibrinogen In those patients who develop a coagulation defect, postmortem intravascular fibrin deposition is only rarely seen Perhaps intravascular coagulation may be caused by severe hemorrhage, and in cases of abruption the depletion of clotting factors seems to be proportional to the amount of blood that is lost This mechanism may play some role in those rare patients who develop a coagulation disorder following severe postpartum hemorrhage Many investigators now believe that the intravenous infusion of thromboplastin leads to the liberation of fibrin split products (fibrin degradation products), which act as inhibitors of the conversion of fibrinogen to fibrin Therefore, fibrinogen is destroyed blood cannot clot, and a hemorrhagic diathesis is produced In these cases it has been suggested that heparin will break the vicious cycle and control bleeding Although heparin may be useful in the intrauterine dead fetus syndrome, prompt delivery is a better solution in the patient with abruptio placentae The following case history is instructive

Case Report

The importance of delivery in treating the coagulation defect in abruptio placentae is shown by its effect upon coagulation

A patient who had severe abruptio placentae was delivered within 1 hour of admission Blood was drawn just after delivery for a coagulation profile The patient's fibrinogen level despite the fact that she had a severe coagulation defect, was over 200 mg/100 ml and her platelet count was over 200 000/cu mm Her partial thromboplastin time was over 60 and her prothrombin time was in the region of 30% Factors V VIII and XIII were all low Plasminogen was low and thrombin time was relatively high Fibrin split products were present in large amounts Over the next 24 hours all the factors mentioned returned to normal, and yet this patient received *no* heparin *no* fresh blood and *no* fibrinogen All that was done for this patient was to allow her prompt delivery This may seem old fashioned, but it is still the best method of management

In studying a group of patients with abruptio placentae, Sutton *et al* (1971) reached the following conclusions

1. Patients with abruptio placentae diagnosed antepartum have a high incidence of coagulation disturbances (60% in their series, whereas some textbooks give the figure as low as 2%)
2. Increased systemic fibrinolysis is rare, and when it does occur in obstetric cases it is almost always secondary to intravascular coagulation
3. The bleeding diathesis is due to a consumption coagulopathy

4. Severe bleeding may occur in the presence of normal fibrinogen concentrations, and this is a point of practical importance The decreased coagulability is due mainly to the effects of fibrin split products
5. The most sensitive tests for the diagnosis of intravascular coagulation are detection of fibrin split products, thrombin time, factor VIII level, partial thromboplastin time, Quick time (prothrombin time), and levels of factors V and XIII
6. The defibrination process is self-limited and is corrected spontaneously following removal of the placenta

Thus the modern management of abruptio placentae remains essentially as follows

1. Give oxygen by face mask (6 liters/min)
2. Record hourly urinary output as well as intake of all fluids
3. Replace blood early The amount should be based upon central venous pressure monitoring (or pulmonary artery wedge pressure) if available Pulse and blood pressure recordings alone do not accurately reflect the situation blood replacement based upon them is often inadequate
4. Investigate clotting time, clot retraction, fibrinogen, and hematocrit, as a minimum Get as good a coagulation profile as is available A prothrombin time and a platelet count are also useful and are available in most hospitals
5. If a coagulation defect is present give fresh frozen plasma This supplies factors V and VIII and approximately 2 g fibrinogen per liter If fibrinogen is depleted, give cryoprecipitate (5–25 units)
6. Give fibrinogen (2–6 g) only if other measures fail or if cryoprecipitate is not available Fibrinogen is still useful in a patient with severe abruptio placentae, a demonstrable deficiency of fibrinogen, and an indication for a cesarean section
7. Use heparin only if delivery and the replacement of coagulation factors does not control bleeding It is rarely indicated in abruptio placentae
8. Give epsilon aminocaproic acid (EACA) only if marked fibrinolysis is evident and heparin is used concomitantly (see section on DIC with Obvious Bleeding in Ch 2) The initial dose is 4 g intravenously, and then 1 g every 4 hours as required Remember that EACA will pass to the fetus
9. Remove the source of thromboplastin by emptying the uterus as soon as possible, either abdominally or vaginally In contrast to placenta previa, abruptio placentae in most patients requires vaginal delivery However, with the increased availability of neonatal intensive care units, cesarean section is being used as the method of delivery in an increasing number of cases Amniotomy and/or oxytocin infusion are indicated to induce or augment labor As labor is carefully monitored, it should be terminated by cesarean section if any of the following indications develop

   A. Fetal distress
   B Increasing uterine tone—*e g* , failure to relax between contractions
   C. Increased bleeding
   D. Evidence of coagulopathy
   E. Poor progress in labor

In centers with good neonatal intensive care units the perinatal mortality will probably be reduced by increasing the use of cesarean section in women with moderate to severe abruption (grades 2 and 3) when fetal heart sounds

are present Cesarean section should be performed for the benefit of the mother as well if abruption is severe and delivery within 2–3 hours is unlikely

Comments on Detailed Management of Delivery

The route chosen should be one to suit the individual patient and will depend on several factors The factors influencing the decision are 1) whether the baby is alive, 2) whether there is fetal distress, 3) the state of the cervix, 4) the quality of uterine contractions, and 5) the general condition of the patient The primary aim is to empty the uterus as soon as possible by the safest route for the individual patient Prompt delivery reduces the possibility of defibrination and other complications

When a baby is alive and the delivery is not expected within 1–2 hours, and a definite diagnosis of severe abruptio placentae has been made, cesarean section will give the greatest chance for fetal survival The abdominal route is used for delivery of the primigravida although, in the presence of abruption, even she may deliver with amazing alacrity If delivery seems likely within 1–2 hours, a vaginal delivery should generally be allowed This is more likely to be undertaken in a multiparous patient, particularly when progressive effacement and dilatation of the cervix are occurring

If there are no contraindications, oxytocin (10 units in a liter of 5% dextrose in water) may be given slowly intravenously, starting at 4 mU/min to improve the quality of labor *At the same time, rupture of the membranes should be performed to expedite delivery and to reduce intrauterine pressure* By reducing uterine distention, rupture of the membranes will probably reduce the possibility of the escape of thromboplastins into the maternal circulation and also reduce the possibility of postpartum uterine atony and acute renal failure When the uterus fails to respond satisfactorily to oxytocin stimulation, abdominal delivery should be considered When a vaginal delivery is being allowed, the patient's blood pressure, the degree of cervical dilatation, and the central venous pressure reading should be recorded every 5 min and the fetal heart rate and uterine contractions monitored continuously Meanwhile, supportive measures are continued and facilities for immediate cesarean section should be available

A pediatrician skilled in infant resuscitation should be present at the time of delivery, *for the baby is usually hypoxic and frequently premature When the baby is dead, a greater effort should be made to effect vaginal delivery* Even in this instance, however, when the patient is not responding to antishock therapy and labor is progressing slowly, there should be no hesitation in performing a cesarean section for the sake of the mother, provided concomitant measures are undertaken to maintain the blood volume and to correct clotting factor deficiencies Occasionally, the attendant will be pleasantly surprised at the delivery of a living child despite the apparent absence of fetal heart tones during the course of labor Although surgery is sometimes necessary as the *most rapid means of halting the process in a patient with severe abruptio placentae, it is generally undesirable when fresh blood or fresh frozen plasma is lacking* All patients with moderate (grade 2) or severe (grade 3) abruptio placentae should be delivered within 6 hours of the onset of abruption

COMPLICATIONS

Postpartum Hemorrhage

The incidence of this complication is doubled in abruptio placentae and increased by a multiple of eight when a demonstrable coagulation defect is present Rarely is hysterectomy required, even in the presence of a Couvelaire uterus At cesarean section the uterus should be left *in situ* even though it shows marked discoloration, provided it shows a tendency to contract following the administration of oxytocics When the patient has toxemia of pregnancy, oxytocics should not be given prophylactically On the other hand, up to 300 units of oxytocin can be infused over a relatively short period of time when severe postpartum hemorrhage occurs Oxytocin is rapidly metabolized, and it should be used in preference to repeated injections of intravenous ergot preparations Rarely, excessive fibrinolysis may play a part in postpartum hemorrhage If bleeding continues, and fibrin degradation products are present, a heparin infusion should be tried If this fails, EACA should be given concomitantly If postpartum hemorrhage continues, hysterectomy or bilateral hypogastric artery ligation should be performed It is important not to delay the surgical approach until too late

ICF (DIC)

This is probably present to some degree in all cases of severe abruptio placentae and can be detected in about two-thirds Clinical manifestations may occur, such as bleeding from a venipuncture site, petechiae, ecchymoses, or bleeding from the gums, gut, or urinary tract The treatment has already been discussed

Amniotic Fluid Infusion

This is fortunately a rare occurrence In the few instances in which the patient survives this complication, she is treated for nonhemorrhagic shock, provided that replacement of blood loss has been adequate Defibrination may be observed in surviving patients As soon as the diagnosis is made, the patient should be treated with heparin

Rupture of the Uterus

A rupture may occur in association with abruption but is unlikely unless oxytocin has been given to improve labor when there is an unrecognized malpresentation The fundal height should be marked when the patient is admitted, and any increase in uterine size noted because uterine distension may lead to rupture, especially if the patient has a cesarean scar

Postpartum Endometritis

If there is evidence of endometritis (a fairly frequent occurrence), cervical smears should be obtained for culture, sensitivity studies should be made, and the appropriate antibiotic given In the presence of clinical signs of infection an antibiotic may be given on the basis of a Gram stained smear of the cervical discharge without waiting for the antibiotic sensitivity report Select the anti
iat seems to be appropriate on the basis of the Gram stain smear Give antibiotic intravenously so that high levels are obtained at an early stage, as

endometritis generally responds poorly to antibiotics In severe endometritis, an empiric combination of ampicillin, gentamycin, and clindamycin (or chloramphenicol) should be used

Acute Renal Failure

Abruptio placentae is the most common cause of acute renal failure in pregnancy, which occurs in 1 2–3 9% of cases, and is due to acute tubular necrosis or bilateral renal cortical necrosis The severity of the renal lesion is directly related to the severity of abruption (see Ch 5, Acute Renal Failure as a Complication of Pregnancy)

Acute Pituitary Necrosis (Sheehan's Syndrome)

This disorder develops in a very small number of patients suffering severe abruptio placentae or postpartum hemorrhage It occurs especially after prolonged shock Hormonal replacement therapy is the basis of management in these patients

Hemorrhagic Shock

The use of Type O Rh-negative blood can be life saving if matched blood is not available (see Ch 3, Shock)

PROGNOSIS

Currently the maternal mortality of abruptio placentae is less than 1%, but the perinatal mortality remains between 20% and 80% in most reported series of cases In early cases the results will generally be good In late cases the results will be poor, irrespective of experience and methods of management In reviewing the literature, it seems clear that some series are overloaded with early cases and others with late cases Women who have had a severe abruption are prone to recurrence, so they should be advised against further pregnancies

RUPTURE OF THE MARGINAL SINUS

It is now generally recognized that this represents a painless marginal abruption of the placenta (grade 0) It is numerically a common cause of the third trimester hemorrhage The patient presents with vaginal bleeding in which pain is either absent or minimal She is usually in labor, and the presenting part is well down in the pelvis In about 50% of the cases the labor is premature Rarely does the patient require blood transfusion The diagnosis is usually made only after delivery of the placenta (Figs 8-15 and 8-16) On occasion the diagnosis can be made by ultrasonography (Fig. 8-17 and 8-18)

RUPTURE OF THE UTERUS

Rupture of the uterus remains one of the most dangerous complications of pregnancy It is the cause of about 5% of maternal deaths in the United States and is an even greater problem in the underdeveloped countries Many babies

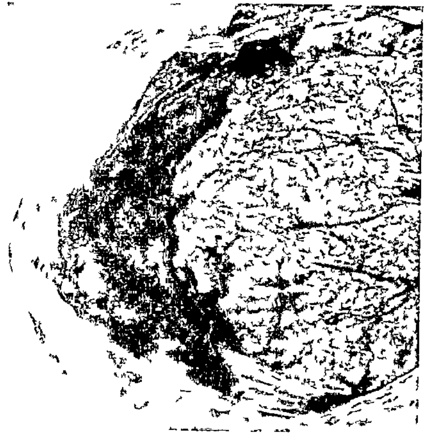


FIG 8 15 Rupture of marginal sinus (Marginal abruption) Typical example with clot in s nus placental surface appears normal (Ferguson JH Hatton RL Am J Obstet Gynecol 78 947 Copyright 1959 American Med cal Associat on)

are born prematurely when rupture occurs so that even if they are born alive their chance of survival is reduced Reported occurrence rates of ruptured uterus vary from 1 137 to 1 4460 Our comments here are based on a review of the literature and on 41 cases seen over a 10-year period during which approximately 50 000 deliveries occurred an incidence of 1 1200

CAUSE

Rupture is designated complete when it extends through the entire uterine wall into the peritoneal cavity or incomplete when the visceral peritoneum remains intact Vaginal bleeding is more likely to be present when the rupture involves the lower uterine segment but most of the bleeding is intraabdominal

INCIDENCE

The condition occurs once in about 2 000 pregnancies

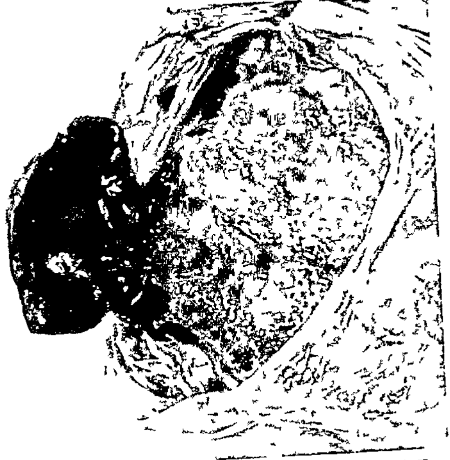


FIG 8 16 Rupture of the marginal sinus (Marginal abruption) Typical example with clot in sinus placental surface appears normal (Ferguson JH Hatton RL Am J Obstet Gynecol 78 947 Copyright 1959 American Medical Association)

CLASSIFICATION

Although here we are concerned with antepartum bleeding the following classification suggested by Eastman is useful from a clinical standpoint

Rupture During Pregnancy

1) SPONTANEOUS RUPTURE OF

a) Previous cesarean section scar
b) Previous operative scar (*e g* myomectomy)
c) The intact uterus Spontaneous rupture of the normal uterus may occur but this is very rare It may occur when deep cervical laceration or adenomyosis of the cervix is present.

2) TRAUMATIC RUPTURE OF THE UTERUS This may occur during over vigorous attempts at external version abortion or, much more commonly, as the result of the injudicious use of oxytocin (Pitocin)

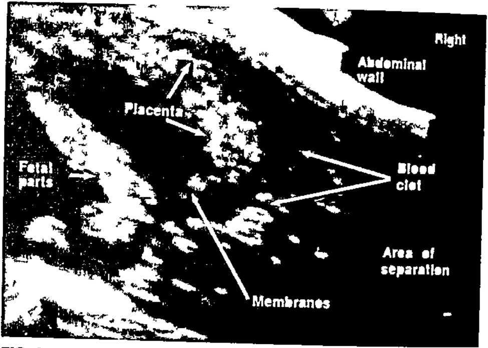


FIG 8 17 Sonogram showing partial placental abruption at lateral edge of placenta with revealed bleeding

Rupture During Labor

1) SPONTANEOUS RUPTURE OF

a) *Previous cesarean section scar* This is more likely to occur in women who have had a "classical" cesarean section (Fig 8 19) and who had their previous cesarean section for placenta previa It is least likely to occur if the previous section has been of the low transverse type The presence of a malpresentation minimum cephalo-pelvic disproportion and a pendulous abdomen all predispose to uterine rupture in labor

b) *Previous operative scar* (e g myomectomy)

c) *The intact uterus* This will usually be seen in cases of obstructed labor

2) TRAUMATIC RUPTURE OF THE UTERUS In this group the commonest causes are the injudicious use of Pitocin the performance of internal version where it is contra ndicated and lacerations inflicted by instruments at the time of delivery

DIAGNOSIS

1) History

When a woman in the latter part of pregnancy or labor develops sudden abdominal pain with nausea and vomiting and shock develops progressively th s condition should be suspected If the patient has had a previous cesarean sec

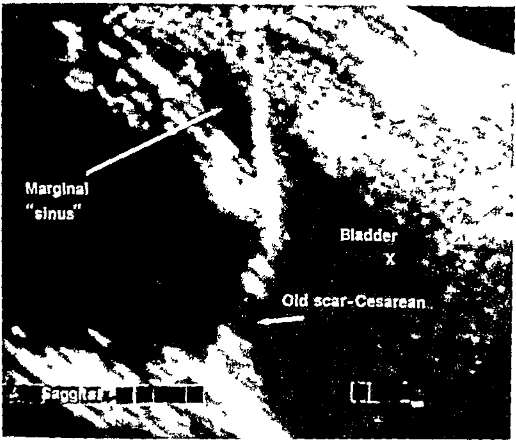


FIG 8 18 Sonogram showing intact marginal sinus or vascular channel in anteriorly located placenta with thinned scar from previous cesarean section

tion, and a malpresentation or a degree of cephalo-pelvic disproportion is found the likelihood is great indeed Pitocin administration and obstructed labor are a catastrophic combination The classic description of 'tearing' pain occurring during labor and followed by sudden cessation of contractions is rather uncommon

When first seen the patient is often in hemorrhagic shock Some vaginal bleeding is usually present but this is more marked when the laceration involves the cervix and lower uterine segment In addition to severe abdominal pain, shoulder pain *may occasionally be present due to the collection of blood under* the diaphragm

2) Physical Examination

This reveals hypotension and a rapid pulse rate The patient's skin is cold, clammy and pale with cyanosis and air hunger being present in the more advanced cases

Abdominal examination reveals generalized tenderness with "rebound" An abdominal mass is present in which fetal parts are often easily felt The

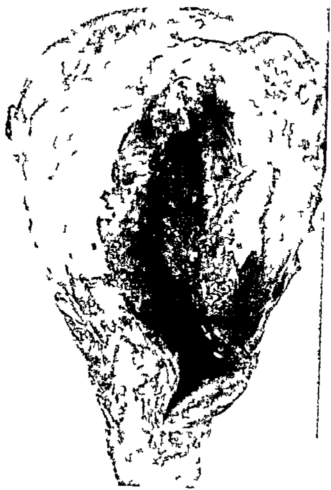


FIG 8 19 Ruptured class cal cesarean sect on scar Th s type of inc s on is undes rable and should rarely be used (Cavanagh D Talisman MR Prematurity and the Obstetrician Englewood Cl ffs Prentice Hall 1969)

abdomen is d stended and dullness is present in the flanks The fetal heart tones are usually absent Vaginal examination may reveal *ascent* of the presenting part as compared w th a prev ous examination

A silent rupture may be found on manual exploration of the uterus following vaginal delivery

PREVENTION

Rupture of the uterus is largely an avoidable acc dent The following steps may be taken to reduce the incidence

1) Repeat cesarean section should be carried out at about the 38th week of pregnancy after careful cl n cal and laboratory assessment of the fetus to

ensure that it is mature About two thirds of the ruptures of cesarean section scars occur after the 38th week of pregnancy

2) Malpresentations and malpositions should be detected at an early stage of labor so that the thinning of the lower uterine segment associated with long labor in these cases may be avoided

3) Pitocin should only be used for the induction or stimulation of labor after the obstetrician has ensured that no contraindication to its use is present

4) Version procedures should be carried out with care Most ruptures occur in association with internal version This should never be carried out unless a definite indication is present The procedure should never be attempted where uterine relaxation is inadequate or when the uterus is retracted around the fetus Cesarean section is a far safer procedure in these cases

In many cases the uterine rupture which occurs during labor is not detected until it manifests itself by postpartum hemorrhage In an effort to recognize rupture of the uterus at an early stage, a vaginal speculum examination should be carried out after every delivery so that cervical tears may be detected and examined After every difficult delivery the uterus should be explored so that lacerations of the lower segment may be detected prior to the development of a broad ligament hematoma

MANAGEMENT

In essence the management of rupture of the uterus during pregnancy and labor consists of the following steps

1. Give adequate blood transfusion and treat hemorrhagic shock
2. Explore the abdomen as soon as possible If uterine rupture is suspected, no time should be wasted in trying to get the patient "in condition" for the operation, otherwise the operation is liable to be a post mortem If anatomic landmarks are obscured by rapid bleeding when the abdomen is opened, the aorta should be located and compressed just above the bifurcation An assistant can control hemorrhage by aortic compression against the vertebral column either manually or using a 'sponge stick,' so that the surgeon is free to deal with the problem at hand This simple procedure of aortic compression can be used in any situation in which massive bleeding in the pelvis obscures the anatomic landmarks, and its use will avert some of the complications of blind clamping, such as damage to the ureter

If the baby has been extruded through a complete rupture, it is first removed When the laceration in the uterine wall is small, an attempt at repair may be made in a young patient, but generally hysterectomy is required Total hysterectomy is usually the procedure of choice, and was performed in 29 of our 41 patients The decision on this will depend upon the condition of the patient and the experience of the surgeon Because palpation of the cervix is difficult, it is usually better to remove the corpus, and then to remove the cervix separately after a longitudinal incision has been made downward through the cervix into the upper vagina This action effects adequate visualization of the entire cervix

Subtotal hysterectomy (performed in 4 of our 41 patients) may not stop bleeding if the rupture involves the cervix If the cervical stump has to be left, postoperative vaginal bleeding may be controlled by suturing the cervical laceration from below and by tight vaginal packing

There is no place for expectancy in the management of a patient with a ruptured uterus It cannot be overemphasized that when a definite diagnosis of ruptured uterus has been made, immediate laparotomy is essential, regardless of the presence of shock It is for emergencies such as this that type O-Rh negative blood should be immediately available to every Labor Delivery Unit. The blood should be crossmatched quickly and given promptly, so that the patient will not bleed to death while prolonged grouping and matching tests are being performed In patients with catastrophic obstetric hemorrhage, the dangers of incompatible blood transfusion from rapid matching techniques must be accepted as a calculated risk

If complete uterine rupture occurs at home, the possibility of maternal survival is inversely proportional to the time taken for transfer to the hospital When the rupture occurs in a large, well equipped hospital, the mortality

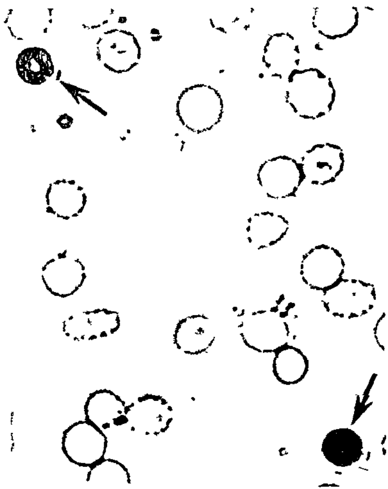


FIG 8 20 Cord blood A Kleihauer Betke stain technique demonstrates erythrocytes with fetal hemoglobin as dark cells (arrows) whereas the erythrocytes with adult hemoglobin appear as ghost cells (Queenan JT JAMA 191 943, 1965)

FIG 8 21 Velamentous insertion of cord may be associated with vasa previa (Ferguson JH Hatton RL Am J Obstet Gynecol 78 947 Copyright 1959 American Medical Association)

should be less than 1% Particularly in rural areas with poor hospital facilities most maternal deaths associated with uterine rupture result from the injudicious use of oxytocin and from internal version attempts when rupture of the membranes has already occurred When complete rupture of the uterus occurs there is little chance of obtaining a live baby The best chance for fetal survival lies in early diagnosis and prompt abdominal or vaginal delivery When rupture of the uterus is suspected preparations should be made in the nursery to receive the depressed and frequently premature baby

VASA PREVIA

This condition does not endanger the life of the mother but the fetal mortality is high The diagnosis should be suspected before delivery if fetal distress occurs with vaginal bleeding. Normoblasts or fetal hemoglobin are found in specimens of the vaginal blood (Fig 8 20) Usually however the diagnosis is made only after examination of the delivered placenta because the condition is most frequently found with velamentous insertion of the cord (Fig 8 21) When

an intrapartum diagnosis of vasa previa is made and the baby is alive, cesarean section should be performed immediately unless vaginal delivery is imminent Only in this way can fetal salvage be obtained This condition is usually encountered following spontaneous rupture of the membranes, but it should be kept in mind when membranes are being ruptured artificially

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Chapter 9

Intrapartum Emergencies

Timothy C. F. O'Connor, Denis Cavanagh

Logical consequences are the scarecrows of fools and the beacons of wise men
T H Huxley (1825–1895)
Animal Automatism

PROLONGED LABOR AND DYSTOCIA

It has been known for many years that the longer labor progressed, the more hazardous became the plight of both mother and infant Prolonged labor was associated with a marked increase in maternal infection and hemorrhage (Fig 9-1), in addition, it had a shattering effect on the morale of the mother and her attendants The fetus fared no better (Fig 9-2)

Early attempts to shorten desultory labor, which varied from "accouchement forcé" and Duhrssen's incisions to forceps extraction through an incompletely dilated cervix, though no doubt well intended, often ended in disaster Thus, the attitude of the obstetrician toward labor became one of masterly inactivity Frequently, medical aid consisted only of hydration and sedation until the eventual distress of all concerned (mother, fetus, physician, and nurse) led to cesarean section or a difficult forceps delivery The principal reasons for this were a reluctance to repeat earlier mistakes, an inbred fatalistic attitude toward labor, and an inadequate understanding of the normal and abnormal patterns of labor Recent developments have led to a reevaluation of this traditional approach, and it is now possible to prevent prolonged labor without placing the mother or baby in jeopardy

Slow progress in labor may be caused by faults in the powers (dysfunctional labor), the passages (contracted pelvis), or the passenger (malpresentations or congenital anomalies), or any combination of these Malpresentations and major congenital anomalies can be easily diagnosed, but often there is great difficulty in trying to differentiate between fetopelvic disproportion and poor uterine action as the cause of the delay The scheme of active management of labor as outlined below is a major advance in the management of this difficult problem

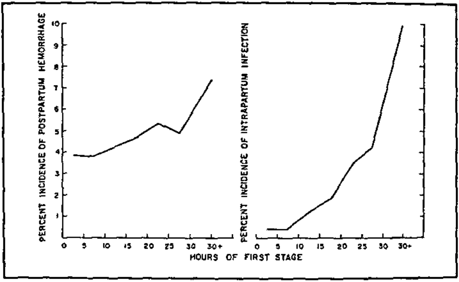


FIG 9-1 Effect of duration of first stage of labor on maternal hemorrhage and infection Both postpartum hemorrhage and infection are more common following prolonged labor (Adapted from Hellman I M, Prystowsky H Am J Obstet Gynecol 63 1223, 1952)

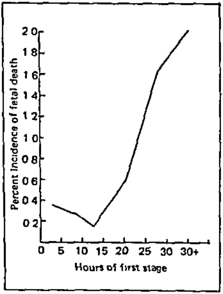


FIG 9 2 Effect of duration of first stage of labor on infant mortality Infant mortality rises steeply after 18 hours in the first stage of labor (Adapted from Hellman LM, Prystowsky H Am J Obstet Gynecol 63 1223, 1952)

The obstetric population may be conveniently divided into two groups, primigravidas and multiparas The former are most prone to develop difficult and prolonged labor, and we shall therefore address most of our remarks to problems met within this group However, it must be emphasized, from the beginning, that multiparous patients in some important ways behave quite

differently from primigravidas in labor, and the scheme of active management outlined *cannot* be applied to multiparas without modification This aspect will be explored further later

BACKGROUND

For many years prolonged labor was defined as a duration of more than 24 hours, hence the maxim—"Never let the sun set twice on a laboring woman" Not until Friedman published his pioneering graphic studies did it become apparent that this was far too generous a time span He demonstrated that the curve of labor obtained by plotting duration against dilatation of the cervix showed a characteristic sigmoid pattern

He divided the first stage of labor into a latent phase and an active phase (Fig 9-3), followed by the traditional second and third stages of labor The *latent phase* is a phase of slow dilatation, which lasts from the onset of labor to the commencement of the active phase at 2–3 cm dilatation He found the mean length of this phase of labor to be 8 6 ± 6 hours in the primigravida and 5.3 ± 4 1 hours in the multipara The **active phase** is one of much more rapid progress, culminating in the start of the second stage The mean length of this phase is 4 9 ± 3 4 hours in the primipara and 2 2 ± 1 5 hours in the multipara

This graphic description helped dispel many of the myths about labor and placed the treatment of dysfunctional labor on a more logical basis Friedman's graph has been used as a basis for the construction of partograms to give a visual and more accurate picture of progress in labor (Fig 9-4) Friedman's original work was retrospective and took zero point on the abscissa as the time of onset of contractions In practice, however, this event is so variable that many workers have found it to be of little value for prospective analysis They selected time of admission to the hospital as a more accurate point from which to start If the findings of workers who took Friedman's starting point (Fig 9-5) are separated from the findings of those who took admission time as their starting point (Fig 9-6), the two curves differ in shape The curves resulting from the findings of those who followed Friedman's format show a latent period slope, whereas the graphs of the others do not demonstrate any significant latent phase Hendricks, Brenner, and Kraus graphically depicted dilatation of the cervix in the last weeks of pregnancy and during labor (Fig 9-7). The similarity of this graph of "pre-labor" to Friedman's latent phase is striking It may well be that the inclusion of patients who were not in labor at the time of admission influenced the shape of Friedman's original curve

A variety of partograms are now available These are graphic depictions of labor of varying complexity, the central point of which is the graph of cervical dilatation versus time Other parameters that have been added (descent of the presenting part, timing of sedation, etc) are very helpful, but cervical dilatation is the event of central importance

Recent workers attribute the origin of their partograms to the work of Philpott and Castle Because of the nature of their practice, Philpott and Castle needed a simple method of ensuring that patients with developing problems in labor be recognized early and be referred to a central unit They devised a partogram (Fig 9 8) with two diagonal lines designated **alert line** and **action line**. The alert line was designed to separate efficiently the majority

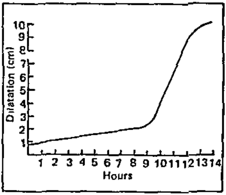


FIG. 9-3. Cervical dilatation as related to time span of labor First stage of labor may be divided into an initial gradual slope of dilatation (latent phase) followed by a steep slope of dilatation (active phase). (Adapted from Friedman EA: Obstet Gynecol 6.567, 1955)

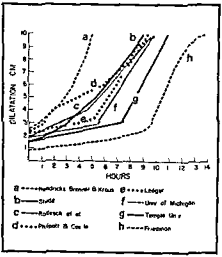


FIG 9-4 Progress of primigravid labor as determined in eight studies Hendricks, Brenner, and Kraus (a), Philpott and Castle (b), Studd (c), Rodesch *et al* (d) Ledger (e), University of Michigan (f), Temple University (g) Friedman (h)

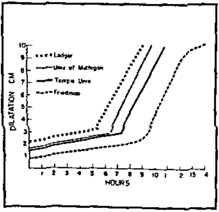


FIG 9-5 Progress of primigravid labor from onset of regular contractions When starting point (origin) on abscissa is taken to be the onset of regular contractions, a latent phase occurs, similar to that shown in Figure 9-3

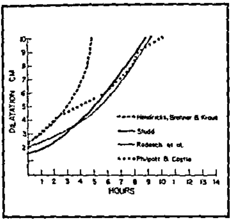
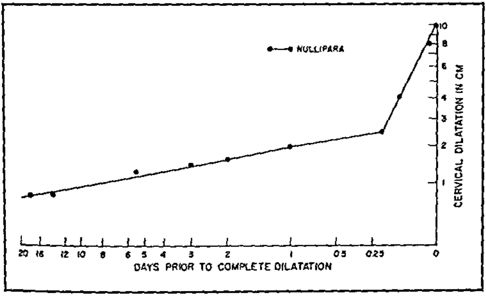


FIG 9-6 Progress of primigravid labor from time of admission When the starting point (origin) begins with admission of patient to hospital no latent phase is observed

FIG 9-7 Slow cervical dilatation quite similar to latent phase of labor, which takes place in late pregnancy (Adapted from Hendricks CH, Brenner WE, Kraus G *Am J Obstet Gynecol 106 1065, 1970*)



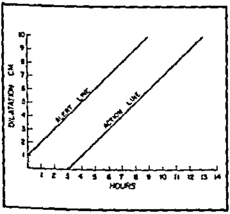


FIG 9 8 Double-line partogram Patients who cross alert line are candidates for high risk obstetric care (Adapted from Philpott RH, Castle WH J Obstet Gynaecol Br Commonw 79· 592, 1972, 79 599, 1972)

TABLE 9-1 Clinical Events in Two Series of Similar Primigravidas

	1966 Series	Present series	Statistical conclusion
Total number	738	624	—
Oxytocin stimulation given	12 3%	9 7%	p<0 01
Labor			
<12 hours	57 5%	94 7%	
>12 to 24 hours	29 5%	4 6%	p<0 001
24 hours	13 0%	0 6%	
Vacuum extraction	9 1%	13 4%	p<0 001
Cesarean section	9 9%	2 6%	p<0 001
Perinatal deaths	5 8%	0 6%	p<0 001

(Adapted from Philpott RH Castle WM J Obstet Gynaecol Br Commonw 79 599 1972b)

of normal from abnormal labors and to allow time for early transfer of the potentially abnormal patient to the central unit following amniotomy If after transfer, the patient's graph of cervical dilatation crossed the second line and there was no contraindication (the action line) labor was stimulated with oxytocin This policy of active management of labor had a dramatic impact on the length of labor, cesarean section rate, and perinatal mortality (Table 9-1)

O'Driscoll and Stronge developed a simple partogram (Fig 9-9) and pursued a similar course of active management Using admission time as zero their results were very similar to those of Philpott and Castle, with the additional observation that instrumental deliveries in primigravidas decreased from 30% to 19 5%, and the need for rotation forceps and difficult extractions virtually disappeared (O'Driscoll, Jackson, and Gallagher)

These investigators and others (Friedman and Studd) have pointed out two problem areas in this simple and otherwise highly successful method of managing labor So long as progress continues to the left of the diagonal line, the labor is classed as normal, so significant delay may not be recognized for some time if the patient has been admitted in advanced labor (Fig 9-10) To avoid this Studd analyzed progress of patients admitted at different degrees of cervical dilatation and converted the data to a labor stencil (Fig 9-11) This stencil can be placed over the partogram and expected progress from different starting points readily observed If dilatation falls 2 hours behind the expected progress, labor is then stimulated

The second problem arises when the patient is admitted with little or no cervical dilatation and the diagnosis of labor is in doubt Three approaches to this group are found in the literature The first is to retrospectively eliminate these difficult cases, which is not helpful The second approach is to treat this group as being in the latent phase of labor and await developments, with or without sedation, thus condemning some patients to an unobserved labor The third approach is that of the "sometimes-wrong but-never-in-doubt" school and entails stimulating some patients who are not in labor Clearly, none of these plans of management is satisfactory

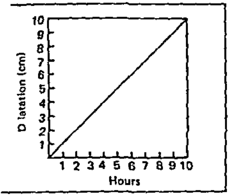


FIG 9 9 Single line partogram. Patients who cross a single diagonal line (as opposed to separate action and alert lines) are managed by amniotomy followed by oxytocin infusion as necessary (Adapted from O'Driscoll K, Stronge JM Clinics Obstet *Gynaecol* 2 3, 1975)

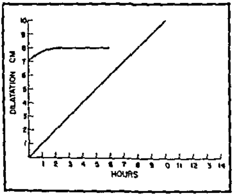


FIG 9-10 Unrecognized secondary arrest This patient would not cross the action line for 7 hours Labor is clearly abnormal

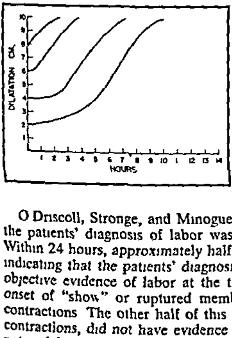


FIG 9 11 A stencil of the type shown may be placed over the partogram Expected progress of labor on admission to the hos pital may be observed at different stages (Adapted from Studd JW Br Med J 4 451, 1973)

O Driscoll, Stronge, and Minogue pointed out that in 10% of primigravidas the patients' diagnosis of labor was not accepted by the admitting physician Within 24 hours, approximately half of these patients were in established labor, indicating that the patients' diagnosis had been correct All these patients had objective evidence of labor at the time of initial admission—*i e*, spontaneous onset of "show" or ruptured membranes in association with painful uterine contractions The other half of this group of patients, although complaining of contractions, did not have evidence of show or ruptured membranes and were not in labor 24 hours later The possibility of error in the diagnosis of labor exists, but if due caution is taken in making the diagnosis in the first place, few

patients will be mismanaged In this study only 45 of 1000 primigravidas spent more than 12 hours in the labor-delivery suite, however of these 45, 38 had been inappropriately stimulated

Careful attention to the diagnosis of labor is necessary since management and outcome will basically depend on this diagnosis If the diagnosis is correct, active management can be expected to yield significant dividends in terms of maternal comfort, safety, and fetal outcome If incorrect, the results can be highly unsatisfactory, as the road chosen will dictate a necessary sequence of events, each of which will compound the initial error

Thus, we have advanced from an initial understanding of abnormal patterns of labor, and an ability to treat them once they are established, to a stage where we can prevent their development In addition to virtually eliminating prolonged labor, a policy of judicial stimulation will diminish the amount of sedation needed, greatly decrease the number of mid-forceps deliveries, and avoid a number of unnecessary cesarean sections

CONTRAINDICATIONS TO STIMULATION OF LABOR

There are two absolute contraindications to stimulation of labor, **malpresentations** (any presentation other than vertex) and **fetal distress.** These must be excluded before labor is stimulated The former may easily be excluded by careful abdominal palpation and vaginal examination, the latter by ensuring that all patients in need of stimulation are afforded the benefit of continuous fetal monitoring Relative contraindications include cephalopelvic disproportion, multiple pregnancies, prematurity, significant bleeding, and multiparity—particularly grand multiparity, which is defined as four or more previous full-term births

The most important of these is multiparity When the primiparous patient develops prolonged labor, it is more often caused by poor uterine action than by true cephalopelvic disproportion Experience with active management of labor has shown that, under proper supervision, the primigravid uterus is virtually immune to rupture

The same cannot be said for multiparous labor, however When the multiparous patient develops prolonged labor, it is very often due to cephalopelvic disproportion, usually fetal in origin and often unrecognized It is imperative, therefore, to eliminate obstruction before stimulating multiparous labor, and failure to do so may result in uterine rupture

PROCEDURE

The partogram is a major advance in the management of labor It is also a very useful teaching instrument

The patient should be examined on admission and a firm diagnosis made as to whether labor has begun Progress should be charted every 2 hours While progress continues to the left of whatever action point is selected (Fig 9 8, 9-9, 9 11), no interference is necessary If, however, the graph shows that the patient's progress is falling to the right of the action line and remaining there for 1–2 hours without spontaneous correction, stimulation of labor becomes indicated Amniotomy and oxytocin infusion are the indicated procedures

Amniotomy

There is now a considerable body of evidence indicating that amniotomy accelerates labor (Kettel and Pettis, 1956, O'Driscoll, Jackson and Gallagher, 1969, Hendricks, Brenner, and Kraus, 1970, Philpott and Castle, 1972, and Studd, 1975) In addition to its stimulatory effect upon labor, amniotomy may also reveal fetal distress signaled by the passage of meconium and is necessary for internal monitoring Amniotomy is rarely indicated before the vertex is engaged, and provided the usual precautions are observed, prolapse of the umbilical cord is no more common following amniotomy than in the general obstetric population (Alderman 1975) The objection is sometimes raised that the obstetrician is committed to delivery after amniotomy, but this argument scarcely applies when the regimen's main objective is to accomplish delivery within a reasonable period of time

Oxytocin

If adequate progress has not developed by 2 hours after amniotomy, the patient should again be assessed to ensure that the vertex is presenting and that there is no fetal distress Then an oxytocin infusion should be commenced at a rate of 1 0 mU/min The rate should be doubled every 15 min until an adequate response is obtained The maximum dose should not exceed 32 mU/min It goes without saying that careful monitoring of uterine contractions and fetal heart patterns is essential

In the rare event that oxytocin stimulation becomes necessary in the multiparous patient, the use of the internal uterine pressure sensing device is advised because of the ever-present risk of uterine rupture It should be kept in mind that hypertonicity may occur at any and all dosages of oxytocin, even one as low as 1 mU/min Positional changes may aid patient comfort, but the left lateral position is recommended insofar as possible, since this position is more hemodynamically desirable, and avoids the vena cava compression syndrome, and in itself often leads to improvement in uterine contractions Occasionally, a distended bladder or rectum is overlooked and labor rapidly improves after the appropriate emptying action has been taken The main limiting factor is the development of fetal distress, and if this should occur, the oxytocin infusion should be discontinued and the patient reevaluated

A small number of patients may not respond as anticipated A possible reason for this is that the initial diagnosis of labor was incorrect If this appears to be the case, a decision must be made as to appropriate further management, usually induction of labor The fact that this situation may occur highlights the importance of making a definite diagnosis of labor prior to the institution of active management Other causes of failure to respond include inadequate delivery of oxytocin or intact membranes despite a history of leakage Either of these two problems is readily corrected Finally, there remains a small group of patients who fail to progress satisfactorily Reassessment often demonstrates that the problem is failure of head descent These are cases of true cephalopelvic disproportion The temptation to "wait a little longer" in such situations should be resisted Such patients should be delivered by cesarean section without delay

PREMATURE LABOR

Prematurity is the greatest single problem in modern obstetrics It is responsible for a large number of neonatal deaths from respiratory distress syndrome and intraventricular hemorrhage It is also responsible for a great deal of neonatal morbidity and subsequent mental handicap Since we do not fully understand the mechanisms involved in the initiation of labor, efforts to stop labor are largely empirical, but a number of approaches have met with some success Nevertheless, there is much conflicting evidence as to the efficacy of the various therapeutic regimens

SELECTION OF PATIENTS FOR INHIBITION OF LABOR

When a patient presents in premature labor, it must first be determined whether it is in the best interests of mother and baby to try to stop labor In many instances any attempt to stop labor will be contraindicated These situations include any serious maternal disease (toxemia, chronic hypertension, hemorrhage, renal disease), or any situation in which the fetus is compromised and would be better off delivered (abruptio placentae, erythroblastosis), or when the fetus is dead or has a major congenital abnormality Spontaneous rupture of the membranes carries with it an increased risk of infection, and most practicing physicians regard this as a contraindication

METHODS OF TREATMENT

The therapeutic regimens which have been proposed to halt premature labor are designed either to decrease uterine response to oxytocin, to decrease prostaglandin production, or merely to decrease uterine contractility

Decreased Response to Oxytocin

It has been thought for many years that progesterone exerted a blocking effect on uterine contractions, and it has been shown that patients in premature labor have low progesterone levels However, all attempts to stop premature labor with progesterone have failed Oxytocin release may be inhibited by rapidly increasing the blood volume, but this is dangerous and only marginally successful in suppressing labor

Intravenous infusion of alcohol was suggested by Fuchs *et al*, as a method of inhibiting oxytocin release It is usually effective initially, but its effect wears off quickly Maintenance dosage may be effective in stopping further contractions but *if discontinued, treatment often has to be restarted* Side effects, such as inebriation, headache, nausea, and vomiting, can be a problem. The danger of aspiration of vomitus must be kept in mind (see Chapter 13)

DOSAGE SCHEDULE

Infusion fluid 100 ml 95% (v/v) ethanol in 900 ml 5% dextrose water = 1000 ml
9 5% (v/v) ethanol (75 4 g/liter)

Loading dose 15 ml/kg body wt/hr for 2 hours
Maintenance dose 1 5 ml/kg body wt/hr for 6 or more hours
Reloading dose If treatment has been discontinued less than 10 hours earlier, the reloading dose is calculated as follows Loading dose × number of hours/10

Decreased Prostaglandin Release

Increased prostaglandin levels in blood and amniotic fluid during labor and also these compounds' ability to induce labor suggest that they play a significant role in human parturition Indomethacin is known to inhibit prostaglandin activity and has been used in at least two trials to abort premature labor (Zuckerman *et al*) Both cite good results but caution that, in animals, prostaglandins play a role in the maintenance of a patent ductus arteriosus, and their inhibition may lead to premature closure and fetal congestive cardiac failure Further study of indomethacin is necessary before its use can be advocated

Decreased Uterine Contractility

Bed rest alone will halt premature labor in 50% of cases, and all claims of therapeutic success must be measured against this yardstick Morphine sulfate is also effective in diminishing uterine contractions but the same is not true of meperidine (Demerol) Diazepam (Valium) is sometimes effective, particularly in the anxious patient However, both these drugs have undesirable effects on the fetus, with their disadvantages outweighing any effect they may have in inhibiting contractions

Beta adrenergic compounds have been found to be effective in stopping uterine contractions both *in vitro* and *in vivo* The various beta adrenergic compounds used for this purpose have the same disadvantages, in particular, cardiovascular side effects (tachycardia and hypotension), but also short duration of effect and tachyphylaxis Because of the cardiovascular effects the patient must be carefully monitored while the drug is being administered

ISOXSUPRINE HYDROCHLORIDE (VASODILAN) This drug was originally tried in oral form, but was found to be ineffective It is now usually given initially by the intravenous route The starting dose should be 500 µg/min, and should not be increased for at least 15 min If contractions continue after this, the dose may be increased, but should not exceed 1000 µg/min Cardiovascular effects are common and may be severe They will usually be apparent after 10 min and may warrant cessation of the infusion, if turning the patient into the lateral recumbent position does not alleviate the hypotension The infusion should be continued for at least 2 hours after contractions cease Thereafter, isoxsuprine should be given by intramuscular injection (10 mg every 4 hours) for 24 hours, and then orally (20 mg every 6 hours) for at least 1 week, and preferably until 36 weeks' gestation has been reached The patient may be allowed home on oral maintenance dosage, provided she restricts her activity However, even with continued maintenance labor frequently recurs

RITODRINE HYDROCHLORDIDE This drug is closely related to isoxsuprine and its effects on uterine contractions have been extensively studied in the United States and Europe Its uterine effects are five to ten times more potent than isoxsuprine, and it has less severe cardiovascular side effects If hypotension and tachycardia do occur, they can usually be corrected by positional change The intravenous infusion can often be stopped after 1 or 2 hours and dosage continued by the intramuscular and oral routes Ritodrine has not been shown to have any undesirable effect on the fetus, but it has been shown to increase uterine blood flow which may be beneficial

Dosage Schedule The initial intravenous dose of 50 μg/min of ritodrine HCl may be increased if necessary but should not exceed 400 μg/min Maintain at the lowest effective dose for 1–2 hours after contractions cease Then intramuscular ritodrine, 5 mg every 4 hours for 24 hours should be given, followed by oral ritodrine, 10 mg every 6 hours Salbutamol is claimed to be a better drug than ritodrine but is not currently available in the United States

OTHER MEDICATIONS Alupent also has tocolytic properties, but tachyphylaxis develops very rapidly, and for this reason this drug has not been used very extensively

In the United States, the most commonly used drugs are alcohol, isoxsuprine and ritodrine They are all effective in the short term but have never been conclusively shown to prolong pregnancy by more than a few days because the diagnosis of premature labor is ill defined and bed rest and sedation by themselves are effective in 50% of cases

Liggins' work with fetal lambs led him to believe that fetal lung maturity could be induced by giving the mother corticosteroids He then used ethanol and salbutamol to stop human premature labor for 48–72 hours, after injecting maternal corticosteroids The decrease in deaths from respiratory distress syndrome in the group of premature infants between 28 and 32 weeks' gestation managed in this manner was most impressive However, some apprehension about the effects of steroids on brain development has been voiced, and further evaluation and follow-up studies must be performed before this very attractive method of management can be recommended for routine use

Management of Premature Delivery

Once it has been decided to allow the premature labor to continue, the following points should be considered

1. The best incubator for the *premature infant is its mother's uterus* If adequate facilities for care of the premature infant are not available to the obstetrician, preparations should be made to transfer the patient, while the fetus is still *in utero*, to a center equipped to manage premature infants
2. Premature infants born in good condition do better than similar infants born in poor condition Recent reports demonstrate that even very small infants do much better when an active approach with fetal monitoring and cesarean section for fetal distress, if necessary, is undertaken In addition to these factors, sedation should be kept to a minimum and regional anesthesia is preferable to systemic narcotics Stimulation of labor is not recommended

3. The premature infant is very susceptible to injury at delivery An episiotomy should always be performed and the head delivered gently between uterine contractions Alternately, elective low forceps delivery may be used to ensure slow, gentle delivery of the head The cord should be clamped and cut immediately and the infant handed to the waiting pediatrician Premature infants are very susceptible to cold injury and should be immediately moved to a heated environment
4. Resuscitation of these infants should be undertaken vigorously when necessary (see Ch 12, *Emergencies in the Newborn*)
5. If the fetal presentation is breech and the estimated fetal weight less than 1500 g recent work seems to indicate that the infant's best chance of survival is delivery by cesarean section

MALPRESENTATIONS

The management of any malpresentation will be influenced by five factors

1. The stage of labor, if the patient is in labor
2. The length of gestation and the estimated fetal size
3. The presence of any complicating factors
4. The particular type of malpresentation
5. The presence of a major congenital abnormality (hydrocephaly, anencephaly, etc)

With these in mind, we will discuss the individual malpresentations in descending order of frequency

BREECH PRESENTATION

The incidence of breech presentations is 3–4% Under ideal circumstances the management of a breech presentation should not be allowed to become an emergency The disposition of each patient should be decided upon before labor starts, but in an imperfect world, this is often not possible

Management

A careful history and physical examination should be carried out with special attention to parity, past obstetric history (if applicable), date of the LMP, estimated fetal size, and pelvic findings All primigravidas, and any multipara who has not delivered a mature infant without difficulty, should have x-ray pelvimetry If there is any doubt about the capacity of the pelvis, cesarean section should be performed It is now well recognized that small breech infants weighing less than 1500 g and large breeches weighing more than 3700 g do poorly if delivered vaginally It is important, therefore, to get an accurate history of the LMP as part of the estimate of fetal size Abdominal delivery for all premature breeches may well become standard practice in the near future Similarly, if there are good grounds for suspecting that the fetus weighs more than 3700 g serious consideration should be given to cesarean section The presence of another risk factor (e g , toxemia, elderly primigravida) would also militate strongly against any attempt to achieve a vaginal delivery A radiograph of the uterine contents should always be obtained to rule out

major bony and central nervous system abnormalities and hyperextension of the fetal head If the latter is noted, vaginal delivery is absolutely contraindicated because of the risk of transection of the spinal cord

If it is decided to allow vaginal delivery in a patient with a breech presentation, the progress of labor must be carefully observed and any significant delay managed by cesarean section rather than stimulation

TECHNIQUE OF BREECH DELIVERY Assisted breech delivery is best carried out under pudendal nerve block anesthesia, but a competent anesthesiologist should be present in the delivery room, if at all possible

There is usually little difficulty if spontaneous breech delivery is allowed as far as possible WAIT—WAIT—WAIT is the watchword It should be emphasized that, particularly in a footling breech, it may be very difficult to diagnose full dilatation, and the patient should not be placed in the lithotomy position until the posterior buttock of the infant is distending the perineum This removes the temptation to pull on the foot, which some practitioners find irresistible Once the patient is in the lithotomy position, a generous episiotomy should be performed, unless the perineum is deficient The patient should be encouraged to push, and the breech should not be touched until the umbilicus has been delivered Such complications as the nuchal position of the arms very rarely occur, except as the result of traction by the obstetrician in an effort to hasten delivery Once the umbilicus has been delivered (and the legs disengaged if necessary) the lower end of the scapula will be visible At this point the arms can usually be delivered without difficulty by hooking the index finger over each of the baby's shoulders in turn (Fig 9 12)

When there is delay in the birth of the **shoulders**, Løvset's maneuver may be used This depends upon the fact that the posterior shoulder has passed through the pelvic brim while the anterior shoulder remains above it By rotating the trunk so that the posterior shoulder is brought to the front, the arm will in the process be pushed downward below the pubic arch and delivered If the trunk is now rotated in the opposite direction, so that the other shoulder is brought into the anterior position, its arm then can be delivered If the arms are in the nuchal position, the situation can be corrected by a similar maneuver, in which the baby is rotated through 180° Thus, in the process of rotation, the anterior arm is pushed forward over the baby's face and eventually downward so that it can be delivered below the pubic arch The same process is repeated for the other arm (Fig 9 13 through 9-17)

The most important aid to successful delivery of the **head** in a breech presentation is the application of suprapubic pressure until the head is well down in the pelvis If the occiput is posterior, rotation to the anterior position is required before delivery of the head is attempted by forceps or by combined shoulder traction and suprapubic pressure

For routine breech delivery, many obstetricians prefer to use Piper or other forceps for delivery of the aftercoming head The forceps should be applied as shown in Figure 9-18 and two assistants should be available at the delivery table

For physicians who are not accustomed to applying forceps in this manner, the method of delivery described by Burns is particularly useful (Fig 9 19

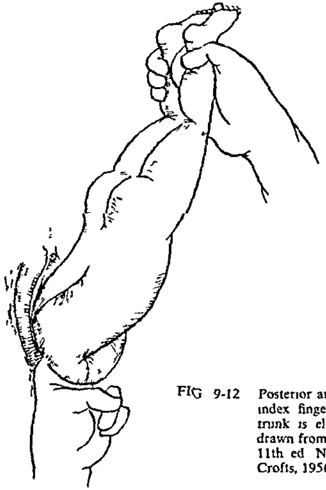


FIG 9-12 Posterior arm is delivered by hooking left index finger over baby's shoulder while trunk is elevated with right hand. (Redrawn from Eastman Williams Obstetrics, 11th ed New York, Appleton-Century-Crofts, 1956)

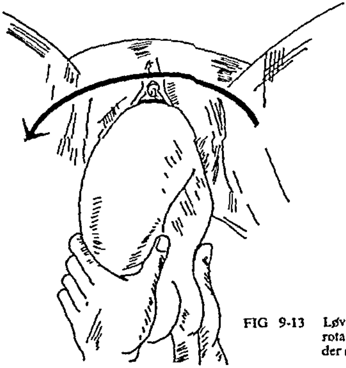


FIG 9-13 Løvset's maneuver Trunk is rotated so that posterior shoulder is brought to front.

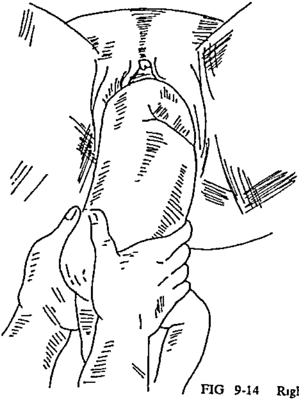


FIG 9-14 Right arm appears below pubic a

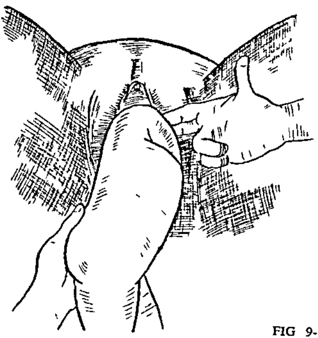


FIG 9-15 Right arm is deliveri

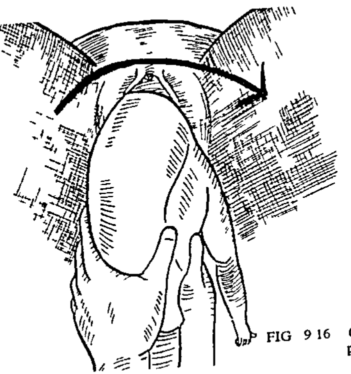


FIG 9 16 Child is now rotated in opposite direction

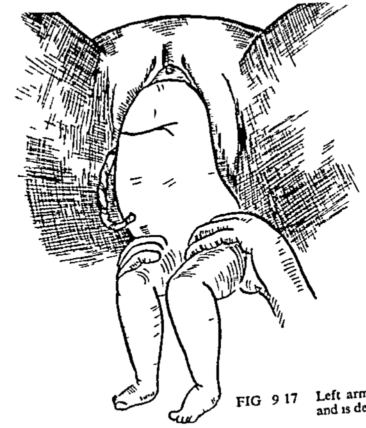


FIG 9 17 Left arm appears below pubic arch and is delivered.

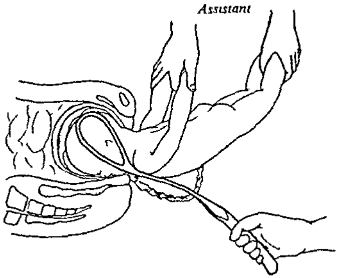


FIG 9 18 Breech Forceps delivery of aftercoming head is carried out while baby's body is held upward by assistant Piper forceps are preferred, and no attempt should be made to apply them until head has descended into pelvis

through 9-22) After the shoulders have been delivered, the baby's trunk is rotated so that the back is anterior The child is then allowed to hang downward over the edge of the delivery table for about half a minute This encourages flexion, rotation and descent of the head

The obstetrician must wait until the suboccipital hairline is visible at the introitus before grasping the child's feet with his right hand Gentle steady traction is applied, downward at first The child's body is then swung upward in an arc, while the traction in the long axis is continued Suprapubic pressure is maintained throughout by an assistant, to facilitate descent and delivery of the head with a minimum amount of neck traction Care must be taken to achieve slow and controlled delivery of the head over the perineum

If this method fails to effect delivery, a modified Mauriceau Smellie Veit technique should be tried (Fig 9-23) The baby is placed astride the physician's left arm, while the trunk is steadied by the index and ring fingers of the right hand placed over the shoulders, the middle finger being extended and resting on the occiput Meanwhile, suprapubic pressure is continued Flexion of the head is effected by applying light pressure with the index and middle fingers of the left hand on either side of the baby's nose, while the middle finger of the right hand exerts pressure on the occiput The baby's head is brought downward until the suboccipital hairline is again clearly visible Flexion and traction are maintained while the trunk is now swung upward, allowing the chin face, and brow to sweep over the perineum in this order As soon as the baby's mouth has been delivered, secretions are aspirated by means of simple bulb suction, thus clearing the airway Delivery of the remainder of the head should proceed slowly over the course of 3 or 4 min, *mainly through the agency of well-controlled suprapubic pressure* applied by an assistant The head must not be

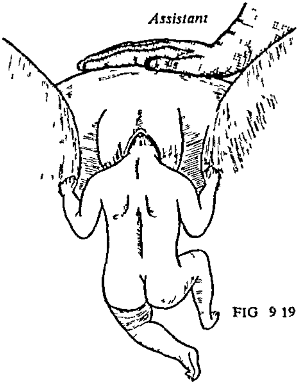
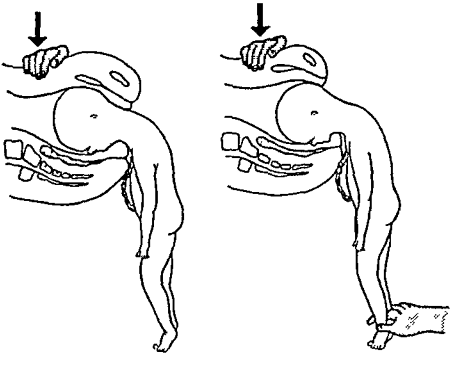


FIG 9 19 Breech Delivery of aftercoming head by Burns maneuver Baby s body is allowed to hang down ward while fundal pressure is applied by assistant

FIG 9 20 When the suboccipital hairline becomes visible below pubic arch, baby's feet are grasped by obstetrician



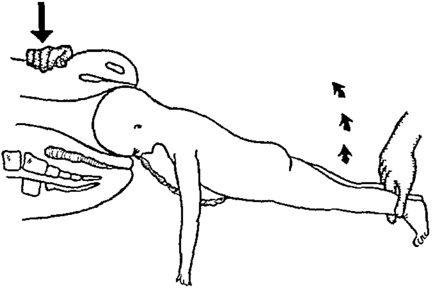


FIG 9 21 After slight initial downward traction child s body is swung upward in an arc Suprapubic pressure is ma ntained throughout by an assistant to minimize neck traction

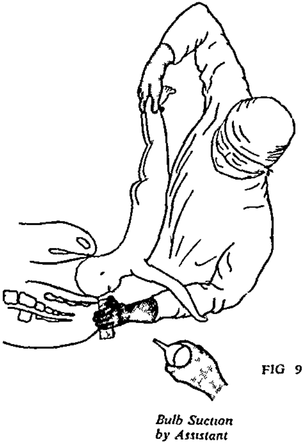


FIG 9 22 Mouth is aspirated by an assistant as soon as pos sible Head is delivered slowly over perineum without aid of supra pubic pressure

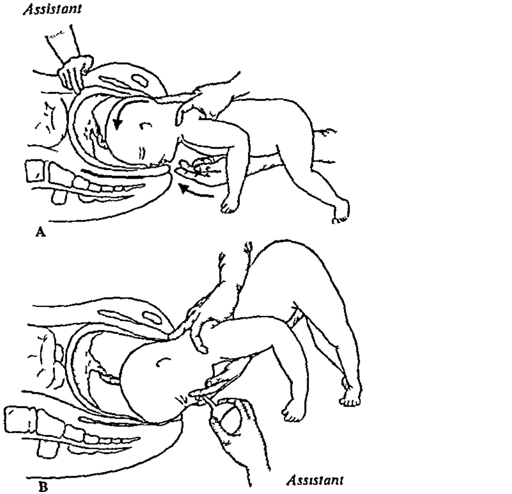


FIG 9 23 Delivery of aftercoming head, using a modified Mauriceau Smellie Veit maneuver **A.** When suboccipital hairline appears below subpubic arch, baby is placed astride obstetrician s left arm Flexion of head is aided by suprapubic pressure by an assistant, as well as by pressure of index and middle fingers of obstetrician's left hand on either side of baby's nose, while middle finger of obstetrician's right hand exerts pressure upward on occiput **B.** Flexion of head is maintained while traction is applied to bring suboccipital hairline once more into view Gentle suprapubic pressure and traction are maintained as trunk is swung upward, allowing the chin, face, and brow to sweep over perineum in this order Baby's mouth and nose are aspirated by an assistant Head should be delivered slowly, 3 or 4 min being allowed

allowed to pop out through the introitus In this respect, bear in mind that about *three times as many breech babies are lost by intracranial hemorrhage from overhasty delivery than are lost from asphyxia*

The presence of a breech presentation makes little difference to the maternal prognosis, but the uncorrected figure for perinatal mortality is much greater for breech than for vertex presentations The factors that contribute to the high mortality in breech births are prematurity, multiple
tal anomalies, prolapsed cord associated with footling

previa However, better selection of cases for vaginal delivery and a more liberal use of cesarean section have greatly reduced perinatal mortality The primigravid breech is so carefully assessed and managed nowadays that it is perhaps timely to emphasize that in many published series of breech deliveries, *perinatal mortality is higher in multiparas than in primigravidas*

FACE PRESENTATION

The incidence of face presentations is 1 600 They are difficult to diagnose by abdominal palpation and, consequently, are not usually recognized until after the onset of labor The diagnosis is usually suspected upon vaginal examination and is confirmed by x ray (Fig 9 24)

FIG 9 24 A Face presentation causing delay in labor Diagnosis suspected clinically and confirmed by lateral pelvimetry film Patient was delivered by cesarean section B Sonogram demonstrating hyperextension of the fetal head (face presentation) in early labor (Courtesy Dr R E Woods)

In early labor, however, a face presentation may be mistaken for a breech presentation, an error that becomes apparent as labor progresses or will be revealed by x-ray pelvimetry Predisposing factors are the presence of a flat pelvis, prematurity, and fetal abnormalities, particularly anencephaly, neck tumors, and fetal hypotonia

In a mentoanterior position, the presenting diameter (submentobregmatic) is no greater than the normal suboccipitobregmatic diameter and is therefore consistent with vaginal delivery However, the presenting part is irregular, and molding of the facial bones is minimal or nonexistent In a mentoposterior position, the presenting diameter is much greater, and vaginal delivery is precluded unless rotation to the mentoanterior position occurs

Management

Trial of labor is quite in order, but the patient's progress should be carefully observed Delay in the first stage should be treated by prompt cesarean section and not by stimulation of labor The presence of some other complicating factor will often lead to early intervention Attempts to rotate the head in mentoposterior face presentation prior to full dilatation are not recommended If delay occurs in the second stage of labor, and the head is in the mentoanterior position, outlet forceps may be used However, between 30% and 40% of mentoposterior positions fail to rotate spontaneously Since they cannot be delivered as such, they will require operative intervention For such cases a number of options have been recommended

MANUAL ROTATION This is often quite easy and a gentle attempt may be tried

FORCEPS ROTATION Forceps rotation with Kielland forceps has been advocated, but it is often difficult, and it may be very traumatic to both mother and infant

RECTIFICATION This maneuver consists of disimpacting the head by pushing it up into the uterus, flexing the head, and bringing it down into the pelvis as a vertex All intrauterine manipulations carry a very real risk of rupture of the uterus and are not recommended unless no other course is open

INTERNAL VERSION AND EXTRACTION This is mentioned only to be condemned Gentle manual rotation in the operating room is the only recommended procedure, and if this fails, the interests of mother and infant are best served by immediate cesarean section

BROW PRESENTATION

The incidence of brow presentations is 1 2,000 Since the diameter presented to the pelvis in brow presentation is the largest of all diameters of the fetal head, vaginal delivery of a term child is virtually impossible Rarely, a very small infant may be delivered as a brow presentation The diagnosis is usually made by vaginal examination and confirmed by x-ray (Fig 9–25)

Management

If the diagnosis is made prior to labor, no action is indicated because spon taneous conversion may occur when contractions start If the diagnosis is made in early labor with intact membranes, and x ray pelvimetry shows a generous pelvis, a short trial of labor may be allowed under constant observation, in the hope that spontaneous conversion to a vertex or face presentation will occur In virtually all other circumstances, cesarean section is the only safe method of delivery Very rarely the patient may be first seen at full dilation, and manual conversion to a vertex or face presentation may be feasible However, it should be emphasized that this maneuver is fraught with danger

TRANSVERSE OR OBLIQUE LIE

A transverse lie (Fig 9-26) is easily diagnosed by abdominal inspection and palpation The most common predisposing cause is the laxity of the uterus and abdominal musculature associated with grand multiparity, but the common association of placenta previa and transverse lie should always be remembered Other underlying causes such as septate uterus, pelvic tumors, contracted pel vis, and prematurity should be borne in mind Fundal implantation of the placenta may be an important factor in etiology, since this will alter the anatomic polarity of the amniotic sac

Management

Placenta previa should be confirmed or excluded by ultrasonic placental localization A careful vaginal examination will rule out pelvic tumors The capacity of the pelvis will often be attested to by the patient's prior obstetric

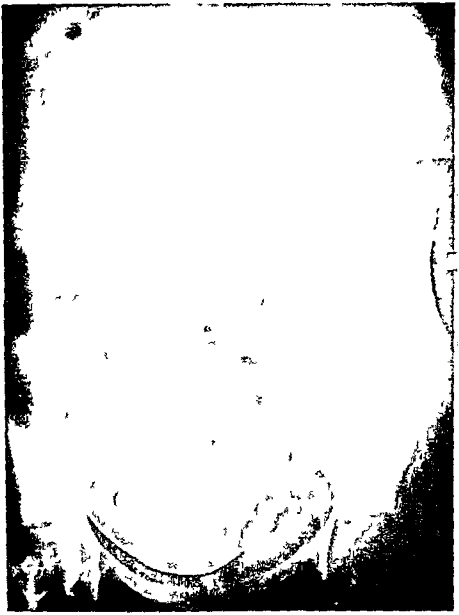


FIG 9 25 Brow presentation causing delay in labor Diagnosis confirmed by anteroposterior film of abdomen Patient was delivered by cesarean section.

performance, but any suspicion of inadequacy warrants x-ray pelvimetry Otherwise, the management of the transverse lie in labor depends on two factors the condition of the membranes, and the stage of labor If the membranes have already ruptured, attempts at version are unlikely to be successful, and cesarean section should be performed without delay If the membranes are intact, particularly in early labor, a gentle attempt at external

FIG 9 26 Transverse lie causing delay in labor Diagnosis confirmed by antero posterior film of abdomen Patient was delivered by cesarean section

cephalic version is indicated This is often possible and if it is successful labor may be allowed to proceed normally but under constant observation as the fetus may revert to a transverse lie at any time When conversion to a vertex or breech is not easy a cesarean section should be performed At operation an attempt should be made to convert the fetus to a vertex or breech so that delivery may be effected by lower uterine segment section rather than by classic section If the patient is infected it is especially important to cover the uterine incision with a peritoneal flap Even if the baby is dead cesarean section is to be preferred to version and extraction or destructive procedures In the

presence of uterine rupture or gross infection, cesarean hysterectomy is often the treatment of choice

DESTRUCTIVE PROCEDURES The older textbooks often recommended that obstructed labor with a dead fetus be treated by destructive operation followed by vaginal delivery This situation arises very rarely nowadays, and most obstetricians have little or no experience with destructive procedures They were designed in an era when cesarean section carried a very high maternal mortality, but even in experienced hands, the procedures were not without risk to the mother Since cesarean section has become a very safe operation, with which every obstetrician is familiar, destructive procedures have very little place in modern obstetrics Two situations in which they may become necessary (impacted shoulders and hydrocephalus) are discussed later in this chapter

THE UNSTABLE LIE

Although not strictly an emergency, the unstable lie is a condition in which, at any time after the beginning of the 38th week of pregnancy, the fetal lie is oblique or transverse, and the presentation varies from day to day This does not include the lie that is fixed in a constant abnormal position Clearly patients in the latter situation are at great risk of prolapsed cord, shoulder presentation, etc, should labor begin outside the hospital For this reason, it has been recommended that such patients be admitted to the hospital to await the onset of labor After admission, the lie will often stabilize, and the patient may then be safely discharged home, unless some other complicating factor necessitates induction However, if the lie does not stabilize, the patient is likely to go beyond term The physician is then confronted with an ugly situation in which the patient, nearly always a grand multipara requires induction of labor if vaginal delivery is to be accomplished

It must be remembered that artificial rupture of the membranes carries a high risk of cord or arm prolapse Furthermore, oxytocin infusion in a woman of high parity with an unstable lie could well result in a ruptured uterus or an amniotic fluid embolus Moreover, 10% of patients with unstable lie have a coexistent placenta previa, and this must be excluded before any interference is contemplated

Management

Very often, if delivery is indicated, the only safe method is by elective cesarean section However, two methods of stabilizing have been advocated for this type of patient, and if unstable lie is a common problem, they may be a suitable alternative to elective cesarean section

The principle behind both the methods described is the same namely, to draw off amniotic fluid after ensuring that the fetal presentation is cephalic and then to induce labor

METHOD 1 Described by Edwards and Nicholson (1969), this approach is claimed to produce very good results The patient is admitted to the labor-delivery unit, an enema is administered and an intravenous infusion of 5%

dextrose in water begun The lie is then checked and, if necessary, external cephalic version performed If the presentation cannot be converted to cephalic, all efforts at induction must be abandoned

Once the lie has been corrected, a fetal heart monitor is connected, and an infusion of oxytocin begun at a rate of 1 mU/min It is important that an experienced attendant remain with the patient at all times, and that the fetal presentation be checked every 5 min Once regular, *not necessarily painful*, contractions are observed, a vaginal examination is performed The vertex presentation is again confirmed and a cord presentation is excluded The membranes are swept off the lower uterine segment, a Drew Smythe catheter inserted, and hindwater puncture performed As much amniotic fluid as possible is drawn off, and the head will usually settle into the pelvis, allowing the forewaters to be ruptured with safety at the end of the procedure Thereafter, the induction may be continued, but the presentation should be checked regularly until the head becomes deeply engaged in the pelvis Meanwhile, the patient should be encouraged to empty her bladder frequently The important step is not to rupture the forewaters until enough fluid has been drawn off and the head has descended into the pelvis The sensitivity of the uterus to oxytocin may alter after amniotomy, so we recommend the insertion of an intrauterine pressure sensor and fetal scalp electrode at time of forewater rupture

METHOD II This approach, described by Ward (1971), also has had good results After placental localization, the patient is admitted to the labor unit, her bladder and rectum are emptied, and if necessary, external cephalic version is performed Amniocentesis is then performed with a Touhy epidural needle, which is connected by transparent plastic tubing to a suction apparatus that produces a negative pressure of about 5 cm Hg The fetal presentation must be checked regularly until as much fluid as possible has been withdrawn The Touhy needle is then removed and an oxytocin infusion is started This is increased from 1 mU/min until regular uterine contractions are apparent and the fetal head has settled into the pelvis The forewaters are then ruptured, and the induction continued with an intrauterine pressure transducer and a scalp electrode in place

MALPOSITIONS

Malpositions are not malpresentations They are normal vertex presentations in which the occiput has failed to rotate into the anterior position The sequence of events in normal labor is that the fetal head engages in the pelvis in the occipitolateral position and, as it descends, rotates to the occipito-anterior position before delivery In about 10% of all vertex presentations, the head enters the pelvis with the occiput posterior Of this 10%, spontaneous rotation to occipitoanterior occurs in 60% and 15% rotate to complete occipitoposterior and are delivered spontaneously in the face-to-pubis position The remainder, 25% of all vertex presentations, require interference

Persistent occipitoposterior position in the multiparous patient is seldom of consequence, because efficient uterine action is usually sufficient to bring

about spontaneous rotation or delivery in face to-pubis position Most problems with occipitoposterior positions arise in primigravidas, in whom it is a common cause of delay in labor

MANAGEMENT

As stated earlier in this chapter, delay in the first stage of labor is common, particularly in primigravidas Delay may be managed by stimulation of labor and this will frequently solve the problem However, a small number of patients will come to full cervical dilatation with the occiput arrested in the posterior, or lateral, position in spite of the active management of labor It has been claimed that the knee chest position encourages rotation of the occiput, but this position is undignified, uncomfortable, and seldom efficacious

Voluntary expulsive efforts may be sufficient to bring about rotation or delivery in the face to pubis position, and frequently the obstetrician can help by gently nudging the occiput in the required direction during contractions If this fails, the obstetrician must review the situation and decide whether further manipulation can be expected to result in an easy vaginal delivery *If any difficulty is envisioned, it is far safer to deliver the infant by the abdominal route Vaginal delivery should never be attempted when part of the head is still palpable per abdomen*

If it is decided to attempt vaginal delivery, three options are open to the obstetrician manual rotation and forceps delivery, forceps rotation and delivery, or vacuum extraction

Manual Rotation and Forceps Delivery

The usual conditions for forceps delivery must be present (see Chapter 10, Emergencies Associated with Manipulative Obstetrics), and general or regional anesthesia is desirable

If necessary, to allow adequate manipulation, a mediolateral episiotomy should be performed before rotation is attempted

If the head is in the right occipitoposterior or occipitolateral position, the *left hand should be inserted into the vagina and the palmar surface of the fingers applied to the occiput while pressure is applied to the anterior shoulder with the right hand Rotation into the occipitoanterior position is carried out,* great care being taken not to push the head above the pelvic brim

It is generally wise to take the rotation about one eighth of a circle beyond the occipitoanterior position so that some allowance may be made for the tendency of the head to slip back to the original position Without removing the internal hand a good cephalic application of the forceps blades should be obtained, and the child is delivered by gentle traction, preferably using an axis traction arrangement

Forceps Rotation and Delivery

This may be achieved with ordinary forceps (Scanzoni maneuver) or with forceps specially designed for the purpose (Kielland or Barton forceps) Resort to these instruments, like the Scanzoni maneuver, is fraught with danger for the physician unskilled in their use

Vacuum Extraction

The vacuum extractor* (ventouse) is particularly suited for delivery of mal positions It can be applied to the most dependent part of the head, and rotation will occur as the head descends and will not be forced It is extremely difficult to exert excessive traction, as this will merely result in separation of the cup from the scalp Furthermore, general anesthesia is unnecessary, and the extensive episiotomy and/or lacerations so commonly associated with forceps rotations are avoided The technique of the use of the vacuum extractor has been described in Chapter 10

MULTIPLE PREGNANCY

The incidence of multiple pregnancy is 1 90 Complications may be already present when labor occurs in a patient with a multiple pregnancy Particularly common are anemia, polyhydramnios, and eclamptogenic toxemia Premature labor is also a common occurrence Early diagnosis is desirable, because extra bed rest reduces the occurrence of the latter two complications All ' large for-dates" patients should, if possible, be referred for ultrasonic examination Since placenta previa is relatively common in association with multiple pregnancy, this is doubly desirable

MANAGEMENT OF LABOR

Although labor is usually normal, overdistention of the uterus sometimes causes uterine inertia Provided the first infant is presenting by the vertex and is not grossly premature, labor may be stimulated at slightly increased risk of uterine rupture, hence, increased vigilance is required In a twin pregnancy, both fetal hearts should be monitored, if at all possible, the leading infant with the scalp electrode, the other with the ultrasonic transducer Delivery of the first infant should be conducted as in any ordinary labor Since the baby is frequently premature, episiotomy should be performed unless the perineum is deficient If the first infant presents by the vertex, spontaneous or low forceps delivery should be the aim If it presents by the breech, an easy assisted breech delivery can usually be anticipated

Delivery of the Second Twin

The second twin is more at risk than the first for several reasons

1. Death of an uniovular twin Death of the second twin may occur unless the umbilical cord is cut between two clamps, since the circulation of the second twin may communicate with that of the first
2. Prolapse of the cord This should always be excluded by pelvic examination immediately after delivery of the first twin
3. Hypoxia The longer the second twin remains in the uterus, the more likely hypoxia is to occur, particularly if more than 15 min elapses between the two deliveries

* Once scathingly described as "peculiarly adapted to a purpose of obstetric surgery namely as a substitute for steel forceps in the hands of men who are deficient in manual dexterity whether from inexperience or natural ineptitude" (Arnott quoted by Chalmers 1963)

1

4. *Uterine inertia This commonly occurs and will contribute to delay in delivery*

Since malpresentations are common in multiple pregnancy and intrauterine manipulation may become necessary, an anesthesiologist should be present, if at all possible Immediately after delivery of the first twin, the lie of the second infant should be ascertained by abdominal and vaginal examination

If the infant presents by the vertex, the head should be pushed into the pelvis and the membranes ruptured If contractions do not restart within 5 min, an oxytocin infusion should be commenced If spontaneous delivery is not imminent after another 5 min and the head is engaged in the pelvis, delivery should be effected by forceps If, however, the head is still high or the cervix has closed down somewhat, vacuum extraction is a much safer procedure than a high forceps operation

If the infant presents by the breech, a foot should be grasped by the examining hand and drawn down into the pelvis Care should be taken to avoid rupturing the membranes until the foot is visible at the introitus Thereafter, the infant may be delivered by gentle breech extraction

If the infant is lying in the transverse position, it can usually be converted to a breech or vertex presentation by external version Should this fail it is quite permissible to perform internal version and breech extraction It is best, when doing this, to grasp the foot through the intact membranes and draw it into the pelvis before rupturing the membranes The intact sac allows the infant to turn more easily, and if the foot slips out of the physician's grasp, the uterus will not clamp down tightly around the infant If fetal distress develops at any time, delivery should, of course, be effected immediately in the most expeditious manner

Very rarely, twins may become locked in the uterus following the delivery of the breech of the first child Whether the infants are alive or dead, if they cannot be unlocked, the safest method of treatment is cesarean section

Postpartum hemorrhage is not uncommon following twin delivery, because the placental site is large, and the uterus often hypotonic Since the mother is frequently anemic, this is a serious complication It is, therefore, wise to have two units of blood available in the event that transfusion becomes necessary Further, an oxytocin infusion (10 units in 500 ml 5% D/W) immediately after the delivery of the second twin, and also ergonovine, unless the patient is hypersensitive. The uterus should be explored manually if any intrauterine manipulations have been performed or if the placenta appears incomplete *Ideally, delivery of women with multiple pregnancies should not be undertaken unless there is ready access to a neonatal intensive care unit*

SHOULDER DYSTOCIA

This condition is usually encountered at the delivery of a large child or an anencephalic fetus

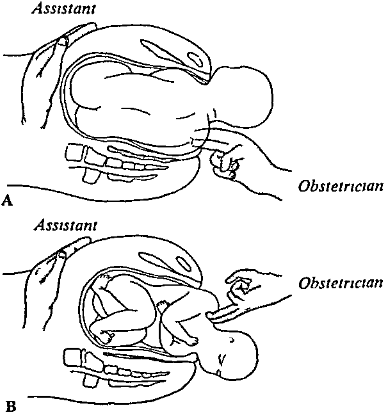
MANAGEMENT

Management of shoulder dystocia may present a considerable problem, and there is usually no time to call for assistance If the head has been delivered and appears normal, the baby's trunk should be palpated with two fingers

in the vagina to check for fetal abnormality such as massive edema If the examination suggests that the child is normal and it is not possible to deliver the anterior shoulder by downward traction combined with fundal pressure, the following procedure should be carried out without delay

1. An episiotomy is performed if this has not already been done, and the vagina is lubricated thoroughly At the same time, an anesthesiologist should be summoned urgently
2. If the patient is awake, she is asked to bear down with her pains while an assistant applies fundal pressure on the uterus At the same time the obstetrician applies pressure with his index and middle fingers over the anterior aspect of the baby's lower shoulder to cause the child to rotate as it descends Usually the shoulder can be delivered after 90° of rotation, but occasionally

FIG 9 27 Shoulder dystocia A Vagina is well lubricated Fundal pressure is applied by assistant while obstetrician applies pressure with his index and middle fingers over anterior aspect of the baby s lower shoulder (Redrawn from Greenhill JP Obstetrics 12th ed Philadelphia WB Saunders 1960) B Baby descends and shoulder can be delivered after 90° to 180° rotation Uterus must be explored after delivery



it is necessary to complete 180° This method was suggested by Wood and is based on the screw principle (Fig 9-27)

3. If this is not successful, general anesthesia should be induced A hand should be inserted into the vagina and an attempt made to deliver the posterior shoulder Should this fail, an attempt should be made to deliver the posterior arm
4. If all these methods fail, the infant will usually be dead by this time, but it is still important to achieve delivery quickly Failure to do so may result in uterine rupture Cleidotomy may therefore become necessary
5. The uterus, cervix, and vagina must always be explored for lacerations following these maneuvers

FETAL ABNORMALITIES

It is sometimes possible to make the diagnosis of fetal abnormality by palpation of the fetal parts The presence of polyhydramnios should increase the suspicion thereof, for in association with this condition, about one third of women have an abnormal fetus An x ray film of the abdomen should be obtained, or preferably, an ultrasonic examination should be performed in all women in whom fetal abnormality is suspected (Fig 9 28 to 9-30) It is possible to detect skeletal abnormalities with a high degree of accuracy on x ray films, although great care should be taken in diagnosing "mild hydrocephalus," particularly when the fetus presents by the breech It is important to diagnose the presence of fetal abnormality during the antepartum period for two main reasons

1. The patient and her family can be prepared psychologically for the possibility that the baby will be abnormal
2. Difficulties in the course of labor can be anticipated and dealt with more effectively should they arise, and the patient should be given adequate sedation during the course of labor

Unfortunately, the patient may be well advanced in the first stage or even in the second stage of labor before the presence of fetal abnormality becomes apparent It is in these cases that the obstetrician may find himself quite suddenly in unexpected difficulty

HYDROCEPHALY

When delay in the birth of the aftercoming head in a breech presentation occurs, the possibility of unrecognized hydrocephalus must always be borne in mind When examination of the suture lines confirms the suspicion, the patient should first be catheterized then a long spinal needle should be inserted through the abdominal wall into the hydrocephalic head By this means, sufficient cerebrospinal fluid can usually be removed to reduce the diameter of the head and allow descent through the pelvis Alternative methods of decompression are to thread a fine catheter up through the meningomyelocele, if one is present, or to perforate the head in the suboccipital region while pulling down on the shoulders

If a hydrocephalic fetus presents by the vertex, the situation is more difficult

◀ FIG 9 28 A X ray film demonstrating anencephalic fetus with polyhydramnios B Anencephalic fetus seen on sonar Spina bifida is also present. (Courtesy Dr R. E. Woods)

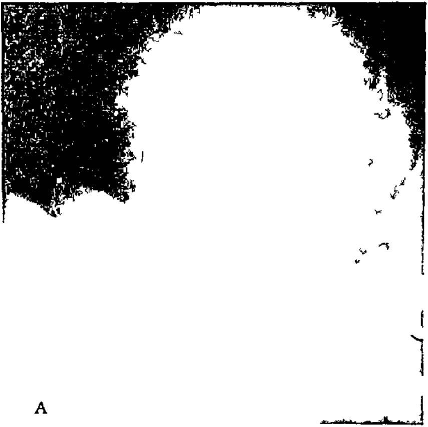
FIG 9 29 Hydrocephalic fetus showing marked separation of skull bones

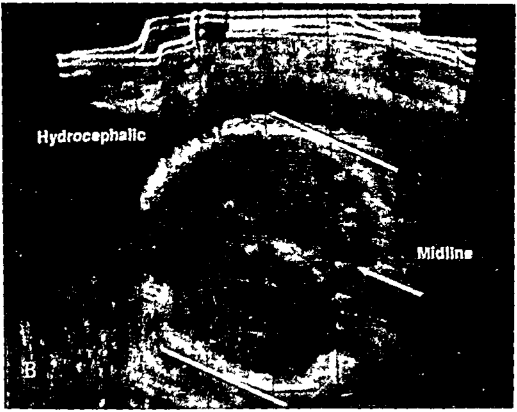
Should decompression become necessary it is safer to do it through the anterior abdominal wall Attempts to decompress the head by the vaginal route are difficult as head is unstable and the needle may slide off and damage the mother However in the presence of gross obesity it may be the only practical route This decompression procedure is safer than craniotomy as the mother is spared the double hazard of lacerations from the craniotomy instrument and the sharp fetal skull bones

ANENCEPHALY

With anencephalic monsters shoulder dystocia is common the largest diameters of the shoulders is much larger than that of the presenting part The patient should be kept well sedated and delivery of the shoulders is not usually unduly difficult because fortunately most of these infants are small If serious difficulty is encountered however Woods method should be used and bilateral cleidotomy should be performed if necessary

FIG 9 30 A Hydrocephalic fetus (lateral x ray view) B Hydrocephalic head (38 weeks gestation) seen on sonar Biparietal diameter (BPD) measures 15 cm (Courtesy Dr R E Woods)





PROLAPSE OF THE CORD

Cord prolapse occurs once in about 300 deliveries It is especially associated with the following situations

1. Prematurity
2. Manipulations such as version and surgical induction of labor
3. A long cord
4. Any cause of poor application of the cervix to the presenting part Mengert and Longwell found prolapse of the cord associated with about 0 4% of vertex presentations, 4% of breech presentations, and 14% of shoulder presentations

Fetal hypoxia results from compression of the umbilical cord between the presenting part and the pelvic tissues The immediate danger to the baby is obvious, and about one third are dead when the diagnosis is made The increased use of cesarean section has increased the fetal survival rate

Remember that the underlying cause of the cord prolapse may endanger the mother's life, and may require intervention even when the baby is dead

MANAGEMENT

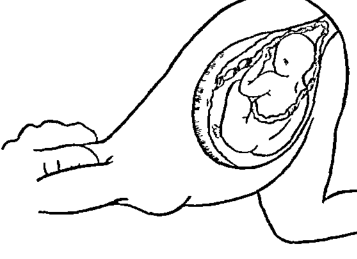
As soon as the patient is seen with a loop of cord in the vagina the following steps should be taken

1. Underlying complications such as tranverse lie which may be dangerous to

the mother must be ruled out An inquiry should be made about vagi bleeding Even as she answers, the patient's abdomen should be palpat rapidly to assess the presentation and position

2. Provided the history and abdominal findings are not suggestive of place previa, an immediate vaginal examination should be carried out
   A. The presence of cord pulsations will indicate that the baby is alive, a the rate and quality will give an indication of fetal prognosis.
   B. The degree of dilatation and effacement of the cervix, and the presen tion, should be determined
   C. Even when no cord pulsation is felt, the fetal heart tones should listened for before the baby is pronounced dead
3. If no underlying complications are present and the baby is dead, then int vention is not required, and the patient is ultimately delivered spontaneou or by low forceps
4. If the baby is alive, its best chance for survival lies in vaginal or abdomi delivery as soon as possible The following steps should be taken immediate
   A. Immediate treatment
      1) The mother should be placed in the knee-chest position (Fig 9-31) reduce the pressure of the presenting part upon the cord If facilit are available, the steep Trendelenburg position is preferred for pati comfort
      2) The presenting part should be elevated by a hand in the vagina
      3) Oxygen should be given to the mother Its administration should continued until delivery, to combat fetal hypoxia
   B. Definitive treatment The definitive treatment is delivery of the infant rapidly and atraumatically as possible The method of delivery will depe

FIG 9-31 Prolapsed cord Knee-chest position reduces cord compression T placement should be combined with administration of oxygen to t mother



upon the degree of dilatation of the cervix and the presentation of the fetus

1) When any presentation other than a vertex or mentoanterior face is present, the treatment of choice is cesarean section The only exception to this is in a multiparous patient with a breech presentation engaged in the pelvis, with the cervix fully dilated, here breech extraction may be preferred If is often very difficult, with a footling breech presenting, to be certain of full dilatation When there is any doubt, it is preferable to deliver the infant abdominally, for if extraction is performed through an incompletely dilated cervix, the cervix will clamp down around the infant's neck, with resulting death or severe damage to the child

2) When the vertex presents, treatment will depend on the dilatation of the cervix If the cervix is fully dilated, forceps delivery is indicated, provided the head is not too high When the cervix is almost fully dilated, it is permissible to try to push the cervix back gently with the next one or two contractions and then apply the forceps An alternative is immediately to apply the vacuum extractor before full dilatation It should be remembered that it takes about 8–10 min to attain an adequate vacuum, and this instrument should not be used if a forceps extraction is feasible If the cervix is not fully or almost fully dilated, delivery should be effected by cesarean section

   Only when facilities are not available for immediate cesarean section should an attempt be made to replace the cord, because excessive handling of the cord may cause spasm of the vessels Replacement is effected by wrapping the slippery loop in moist sterile gauze and inserting the bundle above the presenting part

   Delivery, by whatever route, is a matter of great urgency It was mentioned earlier that one third of infants with this complication are dead when first seen It should also be mentioned that 30% of surviving infants in whom the cord has been prolapsed for more than 30 min will develop cerebral palsy

3) In cord presentation, the cord lies in front of the presenting part, but the membranes are intact If, in the course of labor, fetal distress is evident, this complication should be suspected The diagnosis is confirmed by pelvic examination Great care should be taken during examination not to rupture the membranes, for the presence of fluid reduces the force of cord compression The patient should be given oxygen by mask and placed in a steep Trendelenburg position The fetal heart tones should be monitored carefully If fetal distress continues and the patient is not deliverable from below, the foregoing measures should be continued and cesarean section performed as soon as possible

FETAL DISTRESS

Fetal distress is a loose term that means there are grounds for believing the fetus is in danger of asphyxia Until recently the methods available for assessing fetal well being in labor were very crude, and the diagnosis was un-

satisfactory and often inaccurate Continuous fetal heart rate monitors and fetal scalp pH estimations now help to make the diagnosis more accurate

Traditionally there are three signs of fetal distress convulsive fetal movement, meconium staining of the amniotic fluid, and abnormalities of the fetal heart rate

CONVULSIVE FETAL MOVEMENT

Convulsive movement is often noted by the patient just prior to cessation of all fetal movements It is probably the 'last gasp" effort of the fetus to escape its asphyxiating environment, and since this is a terminal event, it has little or no value in assessing the degree of fetal distress

MECONIUM

The passage of meconium by the fetus is caused by hyperperistalsis of the colon and relaxation of the anal sphincter resulting from hypoxia Postmortem examination of fetuses who have died from intrapartum anoxia almost always reveals an empty colon and terminal ileum Meconium staining of the amniotic fluid is always indicative of fetal hypoxia, be it current, recent, or remote The only exception to this is a breech presentation engaged in the pelvis Careful examination of the meconium can be revealing and is well worth while Freshly passed meconium is bright green in color, and flecks of unstained vernix may be noted in the fluid Old meconium, on the other hand, is less bright in color, and any vernix present will be stained a similar color The volume and consistency of the fluid is also worth noting, as a large amount of lightly stained fluid is much less sinister than a small amount of thick "pea-soup"-like material The latter is due to the passage of meconium into a small volume of amniotic fluid, often associated with intrauterine growth retardation and postmaturity

Although the presence of meconium should always alert the clinician to the possibility of fetal hypoxia, there are a few well-known situations in which fetal hypoxia can occur without the passage of meconium Infants of less than 34 weeks' gestation often do not pass meconium, nor do Rh-sensitized infants with severe anemia Severe fatal abruptions are frequently associated with clear amniotic fluid and, of course the presence of an imperforate anus will preclude the passage of meconium Meconium should always be regarded as significant and at the least an indication for careful fetal heart monitoring

FETAL HEART RATE ABNORMALITIES

Traditionally the normal fetal heart rate is described as being 120–160 beats per minute, with hypoxia causing an initial tachycardia followed by a progressive bradycardia The assessment of the fetal heart rate was formerly performed with the fetoscope, a method both intermittent and subject to observer error The recent advent of the fetal heart-rate monitor has made continuous recording of the fetal heart practical and has eliminated the element of observer error

Three aspects should be noted when assessing the continuous fetal heart rate the basal fetal heart rate, the 'beat-to-beat variation," and the presence of any transient alterations of fetal heart rate

Basal Fetal Heart Rate

The basal rate is the rate between contractions, and it is normally between 120 and 160 beats per minute Baseline bradycardia (Fig 9-32) is defined as a rate of less than 120 beats per minute lasting for over 10 min If the pattern is uncomplicated, it is not an adverse pattern, and no action is warranted However, if baseline bradycardia is complicated by some other abnormality, *it should be investigated further*

Baseline tachycardia is defined as a baseline fetal heart rate of over 160 beats per minute and, if uncomplicated, is not an adverse pattern unless associated with maternal pyrexia or ketosis Baseline tachycardia complicated by *any decelerations may be an early sign of fetal hypoxia* and should be investigated

Beat-to-Beat Variation

One of the characteristics of a normal tracing is a degree of short term variability that reflects central nervous system control of the fetal heart rate This beat-to-beat variation usually ranges from 5 to 15 beats per minute, and good variability is present in a healthy neonate Poor variability (< 5 beats per minute), in addition to being caused by hypoxia, can be caused by a variety of drugs commonly used during labor—*i e*, atropine, tranquilizers, barbiturates, and narcotics Some methods of regional analgesia (epidural and paracervical block) may have a similar effect (Fig 9-33) Baseline tachycardia is commonly associated with an apparent loss of beat-to-beat variation The pattern may be described as uncomplicated if there are no other abnormalities, when it is not necessarily an adverse pattern However, poor variability complicated by an abnormal baseline and/or periodic decelerations is frequently associated with fetal hypoxia

Periodic (Transient) Alterations of Fetal Heart Rate

Periodic alterations are divided into **accelerations** *and* **decelerations** *and are differentiated* from baseline changes in that they last less than 10 min

ACCELERATION PATTERNS *Transient acceleration patterns* (Fig 9-34) *accompany* fetal movements and sometimes may be noted at the start of a contraction They are associated with healthy infants and are not a cause for concern

DECELERATION PATTERNS Three basic types of periodic decelerations may be observed (Fig 9-35) They are usually called **early, late,** and **variable** decelerations

Early Decelerations These are thought to be due to fetal head compression and can be eliminated by atropine They usually start at the onset of the contraction, the nadir of the deceleration coincides with the apex of the contraction, and recovery of the baseline fetal heart rate has occurred by the end of the contraction The magnitude of the deceleration is small, usually less than 40 beats per minute This pattern is generally accepted as innocuous, but

FIG 9-32 Fetal heart rate Basal rate is 110, but there is good variability, and acceleration is noted in addition to mild type I decelerations Infant was born in good condition

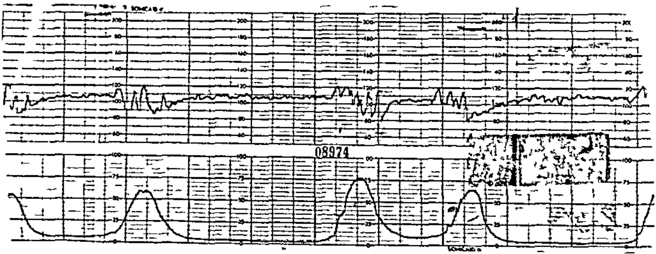
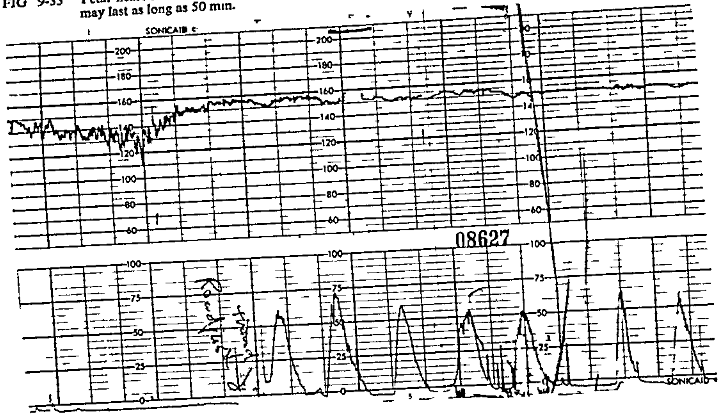


FIG 9-33 Fetal heart rate Paracervical block affects heart rate variability; effect may last as long as 50 min.



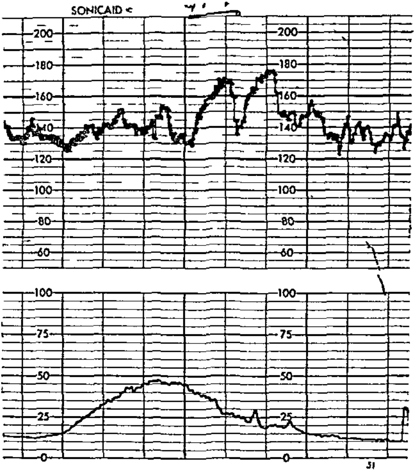


FIG 9 34 Fetal heart rate Acceleration patterns may be associated with contractions or fetal movements In either instance they indicate a healthy fetus

caution is advised, as early decelerations may subsequently develop into variable decelerations (Fig 9 36)

Late Decelerations This pattern of deceleration is caused by uteroplacental insufficiency Late decelerations start after the onset of the contraction The nadir of the deceleration occurs after the apex of the contraction and recovery of the baseline fetal heart rate does not occur until after the contraction is over *This pattern is always significant particularly if complicated by another abnormality* (baseline tachycardia or bradycardia and/or loss of variability)

Variable Decelerations Variable decelerations are said to be caused by compression of the umbilical cord They are characterized by their variable onset They are not necessarily related to uterine contractions They are often U

FIG 9-35 Fetal heart rate Diagrammatic representation of proposed mechanism of deceleration patterns A In head compression pattern (HC), onset of deceleration (arrows) coincides with rise in intrauterine pressure (arrows) Uniform shape of deceleration reflects shape of associated uterine pressure curve B Uteroplacental insufficiency pattern (UPI) is characterized by uniform shape and onset, late in contraction C. Variable deceleration (CC) is of variable shape and does not reflect shape of associated intrauterine pressure curve, its onset is inconsistent in relationship to onset of contraction (Hon EH Atlas of Fetal Heart Rate Patterns New Haven, CT, Harty Press, 1968)

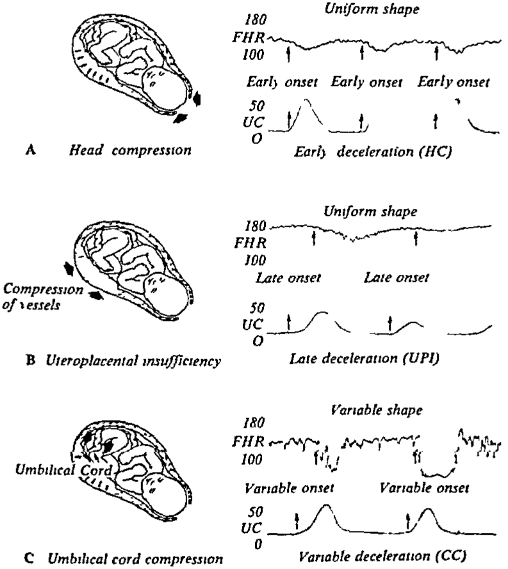
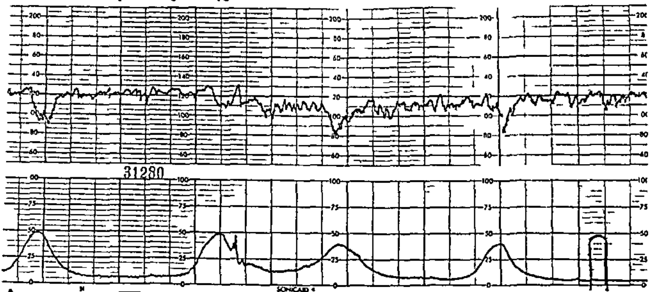


FIG 9 36 Fetal heart rate A Type I decelerations are evident These later develop into variable decelerations B Variable decelerations which disappeared after repositioning and oxygen administration



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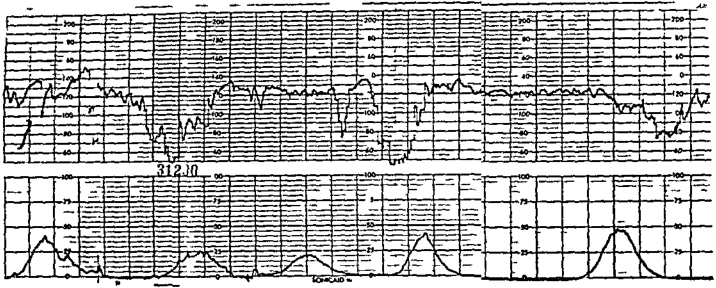

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shaped, with a range difference of over 50 beats per minute. However, this group tends to become a wastebasket into which decelerations that do not fit exactly into the other two groups are placed. When the pattern is uncomplicated (normal baseline rate and normal variability), it is significant but not ominous. However, a complicated pattern (abnormal baseline rate and/or poor variability) should arouse suspicions of fetal hypoxia. All variable decelerations, if consistent, should be investigated.

FETAL BLOOD SAMPLING

Saling first demonstrated in 1962 that it was possible to obtain fetal blood for pH determination. He further demonstrated that measurement of fetal pH blood could be used as a guide to the oxygenation of the fetus in labor. The normal fetus receives adequate oxygen for its needs, but a fetus suffering from hypoxia has to utilize anaerobic glycolysis, the end product of which is lactic acid. When lactic acid accumulates, it leads to a fall in fetal blood pH.

Saling's technique achieved widespread popularity as a method of fetal monitoring prior to the advent of electronic monitoring. However, it has the major disadvantage in that the status of the fetus can be assessed only intermittently. Clearly a normal pH at the present moment is not a predictor of what the fetal status may be in 2 hours' time. Thus, when continuous fetal heart monitoring became available, the technique fell into disuse, until it became apparent that many abnormal fetal heart patterns, while statistically associated with fetal hypoxia, were often not predictive of compromised fetuses in individual patients (Liu and Blackwell, 1975; Thomas, 1975). Most centers now utilize continuous fetal heart monitoring to dictate the timing of scalp sampling; the latter, in turn, helps assess the significance of questionable fetal heart patterns.

Normal and Abnormal Fetal pH Values

During the first stage of labor, the normal fetal scalp pH is in the range of 7.25–7.35. In the late second stage, the mean pH is somewhat lower (7.25). In general, 7.20 is regarded as the point below which acidosis is present, but any level between 7.20 and 7.25 is suspicious enough to warrant a repeat sample. Factors that alter the maternal acid base balance may also alter the fetal pH, and either simulate or mask hypoxia. It is therefore recommended that the maternal blood pH be checked when an unexplained low fetal pH is obtained. Prolonged labor with inadequate hydration predisposes to maternal acidosis, and to "infusion acidosis" in the fetus. Other factors implicated in altering fetal pH are maternal hyperventilation and maternal infusion of Ringer lactate type solutions (Pearson). The validity of scalp capillary blood pH as a reflection of central acid base balance has been established by simultaneous measurement of both in the fetal lamb and monkey (Gare *et al.*, 1967; Abramsons *et al.*, 1968) and by almost simultaneous measurements in the human (Kubli *et al.*, 1966). The formation of caput succedaneum would be expected to cause some fall in pH because of stasis, but both Saling (1967) and Teramo (1969) did not find that this was so.

Technique

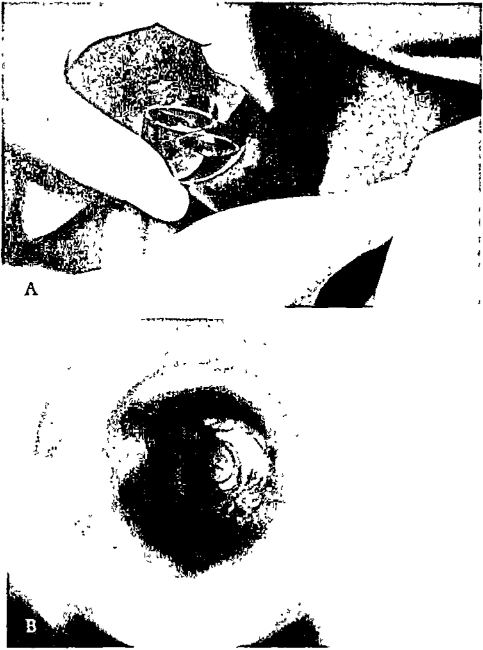
The patient may be positioned in the lithotomy, left lateral (Sims's), or knee-chest position The latter two positions are preferred, as they do not carry the risk of supine hypotension An amnioscope (Fig 9-37A) of suitable diameter, which will be determined by the dilatation of the cervix, is inserted gently into the posterior fornix and the tip guided anteriorly until it is inside the cervix The fetal head is now visualized (Fig 9-37B), and the field cleared of amniotic fluid, blood and debris, with cotton wool swabs Next the scalp is sprayed with ethyl chloride to produce hyperemia The ethyl chloride is cleaned off with cotton wool, and an incision is made at 12 o'clock with the specially designed guarded blade (Fig 9-37C) When a drop of blood appears, it is collected in a preheparinized glass capillary tube A small metal 'flea" is inserted into the tube and agitated with a magnet to ensure thorough mixing of the blood If the sample is not read immediately, it is advisable to seal the ends of the tube with the rubber plugs supplied by the manufacturers of the blood-gas analyzers

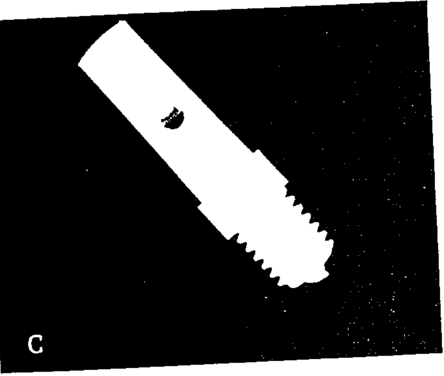
It is important to avoid some of the common sources of error if reliable results are to be obtained 1) Be certain that the field is adequately cleansed, as contamination with maternal blood, amniotic fluid, meconium, or even the ethyl chloride may give misleading results 2) The sample should be taken directly from the scalp and not from a pool of blood which may form at the bottom of the field 3) Lastly, be sure that the analyzer is correctly calibrated and is regularly checked, as recommended by the manufacturer

Complications of Fetal Blood Sampling

Considering the length of time the technique has been in use, the number of reported complications is remarkably small They are, as might be expected, infection and hemorrhage Only four cases of infection, three of the scalp and one of the buttocks have been described (Table 9 2) Hemorrhage from the incision is also rare, a total of only eight serious cases have been described Two of these cases were fatal—one due to the use of an ordinary scalpel blade, which penetrated the fetal skull and lacerated the transverse sinus, the other associated with a severe coagulation defect in the infant Four of the six non-fatal cases were also associated with coagulation defects in the infants There is some evidence that infants who are subject to intrapartum hypoxia develop a coagulation defect of a consumptive type (Chadd *et al*, 1971) Since four of these five infants had low pH readings it is possible that the hemorrhage was a manifestation of fetal hypoxia If the scalp continues to bleed, once the sample has been collected, simple pressure with a cotton wool swab for a few minutes is nearly always sufficient to stop the bleeding but if this is not successful, consideration should be given to delivery In one other case, oozing from the incision was observed at delivery, and the wound was sutured Unfortunately, the suture penetrated the aponeurosis, and the result was a subaponeurotic hematoma If the incision is oozing after delivery, simple pressure will usually be adequate to stop it, but if pressure proves to be inadequate, a small Michel clip would seem to be more appropriate than a suture In the remaining reported case, the vacuum extractor cup was placed over the incision,

FIG. 9-37 Fetal blood sampling. A. Amnioscope is inserted in preparation for taking blood from scalp of fetus. B. Scalp is visualized before sample is taken. C. Specially designed, guarded blade for incision of fetal scalp. Blade is only 2 mm long so that penetration of the scalp aponeurosis will not be permitted.





and the infant subsequently developed a cephalhematoma Accordingly, it would seem wise, in view of this, to avoid positioning the vacuum cup over the site of the incision.

MANAGEMENT OF ABNORMAL FETAL HEART RATE PATTERNS

The management of the patient with an abnormal fetal heart rate will often be influenced by factors other than the tracing alone Factors such as the fetal presentation, the stage of labor, the presence or absence of complicating dis-

TABLE 9-2. Complications of Fetal Blood Samplings

Complication	Cases (No.)	Source
Infection		
Scalp abscess	3	Balfour
Buttocks	1	Kubli
Hemorrhage		
Fatal		
Coagulation defect	1	Beard
Unguarded blade	1	Beard
Nonfatal, serious		
Coagulation defect	3	Kubli (1), Balfour (2)
Vacuum extraction	1	Kubli
"Secondary hemorrhagic disease"	1	Balfour
Sutures	1	Hull and Wilson

orders (toxemia, hemorrhage, meconium) may all influence the management selected

There is general agreement that a normal fetal heart pattern is a reliable indicator of a fetus in good condition An abnormal pattern should alert the physician to the possibility of fetal compromise, but it is often not indicative of fetal asphyxia There is poor correlation between abnormal patterns and fetal acidosis in individual cases, and it can occasionally be difficult to determine which specific abnormality is present Indeed, a number of abnormalities may be present simultaneously, presenting a confusing picture

When a significantly abnormal pattern appears, the first step is to see if it is due to a remediable cause Loss of variability may be caused by recent administration of drugs, and in that case does not usually require any treatment Grossly abnormal patterns may follow epidural or paracervical block, but will usually respond to changes in maternal posture It is always wise to examine the patient vaginally on the appearance of severe variable decelerations to exclude cord prolapse

If no cause for significant abnormalities is apparent, the next step is to alter the maternal position, stop oxytocin (if it is being infused), and give the mother oxygen If, despite these maneuvers, the abnormalities persist for more than a few minutes, a fetal scalp blood sample should be obtained

If this is within normal limits (pH 7 25), the patient may be safely observed, but if the abnormalities persist, or worsen, the sample must be repeated If the pH is in the so-called "gray area" (7 20–7 25), the sample should be repeated in 15–30 min If the pH is acidotic (< 7 20) and there is no evidence of maternal acidosis, the infant should be delivered by the most expeditious route It is not always desirable to wait until the infant becomes acidotic before deciding on delivery, as a falling pH together with persistence of abnormal patterns is ample evidence of fetal distress The combination of late decelerations with loss of variability is ominous enough to warrant delivery without recourse to a scalp sample

Neither of these two techniques is foolproof, but used together they can significantly help the obstetrician make a more accurate diagnosis of fetal distress It must be emphasized, however, that they are ancillary aids that contribute to the quality of clinical judgment but cannot replace it

It is possible that in the future other parameters of fetal oxygenation, such as blood lactate levels, will prove to be more valuable than scalp blood pH estimations

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Emergencies Associated With Manipulative Obstetrics

Timothy C. F O'Connor

Chapter 10

O judgment thou art fled to brutish beasts
And men have lost their reason!
William Shakespeare (1564–1616)
Julius Caesar Act III Scene 2

It is a tragedy indeed when a maternal or fetal death or damage occurs following a procedure for which there was no indication In this chapter a number of procedures commonly undertaken in obstetrics are discussed which, when used wisely, have done much to reduce maternal and fetal mortality

INDUCTION OF LABOR

INDICATIONS

Medical

Any complication of late pregnancy that threatens the welfare of the mother or fetus constitutes an indication for delivery Whether delivery is achieved by induction of labor or by elective cesarean section depends on a number of factors including the presentation of the fetus, the station of the presenting part, and the condition of the cervix In the past, induction was often advocated at an arbitrary time for various disorders that endangered the fetus—*e g*, postmaturity, diabetes, rhesus incompatibility The induction was often carried out under unfavorable circumstances that led to a significant number of failures With the advent of the various methods of assessing fetal well-being, it is now possible to cater for the needs of each fetus, often enabling the deferment of induction until conditions are more propitious

Elective

There is no doubt that when there is good evidence of fetal compromise, delivery is indicated, but stretching the indications for induction to include all fetuses at risk, real or imagined, has not been shown to produce better results in terms

of fetal outcome Similarly good results have been reported in series in which induction was universal (Lampe, 1975) and exceptional (O'Driscoll *et al* 1975) Comparative studies of groups of patients managed by elective or partly elective induction with groups managed more conservatively seem to indicate that there is little or no advantage to the aggressive approach (Chalmers *et al*, 1976, Cole *et al*, 1975) Studies have suggested both that benefits may be derived from a policy of frequent induction (Tipton and Lewis, 1975) and that unnecessary risk may be generated (Liston and Campbell, 1974) O'Driscoll *et al* describe the dilemma well

> There is a subtle influence in obstetrics that operates to absolve a doctor who intervenes in the course of normal pregnancy and which by implication, exposes his conservative colleague to censure for inactivity when an infant is born dead

The most that can be said for elective induction of labor at this time is that it has not been shown to be positively harmful

SELECTION OF PATIENTS FOR INDUCTION

Malpresentations contraindicate induction of labor If a patient with a malpresentation, particularly a breech presentation, needs delivery, the safest way to deliver the infant is by cesarean section The subject of the unstable lie and stabilizing induction has been considered in Chapter 9

When the need for delivery is urgent and the presentation cephalic, induction should be attempted even when the circumstances are unfavorable

However, when the need for delivery is not urgent or when elective induction is being considered, it is wise to assess the chances of success or failure beforehand To this end, a variety of scoring systems have been devised (Bishop, 1964, Fields, 1966, Burnett, 1966, Friedman 1967) These include a number of variables—the condition of the cervix (dilatation, effacement, and consistency), the station and position of the fetal head, estimated fetal weight, and length of gestation The most widely used of these scoring systems is the Bishop score (see Table 10-1) Induction of labor with a poor Bishop score (or other score) should not be undertaken as an elective procedure or when the indication for induction is of doubtful validity

METHODS OF INDUCTION

Until the physiology of the events leading to the onset of normal labor is fully understood, no universally successful method of inducing labor will be devised However, artificial induction of labor is a much more successful procedure than prevention or halting of premature labor

All the methods to be described are successful in the majority of patients, but often the failures, which usually occur in cases with low Bishop scores, occur in the very patients the obstetrician is most anxious to deliver

Artificial Rupture of the Membranes

This procedure alone will be successful in inducing labor within 24 hours in 90% of term pregnancies (Friedman, 1976) It carries with it the inherent risk of amnionitis and infection of the fetus, particularly when the interval be-

TABLE 10-1 Bishop Scoring Index

	Rating			
Factor	**0**	**1**	**2**	**3**
Dilatation	Closed	1 2 cm	3-4 cm	5 cm+
Effacement	0-30%	40-50%	60-70%	80% +
Station	-3	-2	-1 0	+1 +2
Consistency	Firm	Medium	Soft	
Position	Posterior	Middle	Anterior	

Range of scores 0–13

Prerequisites Multiparity gestation of at least 36 weeks and vertex presentation with a normal past and present obstetric history

Predictions Patients with a score of 9 or more will have a safe successful induction with an average length of labor of less than 4 hours (Bishop EH Pelvic scoring for elective induction Obstet Gynecol 24 266 1964)

tween induction and delivery is prolonged Because of this risk of infection, artificial rupture of the membranes is usually combined with oxytocin infusion This combination increases the chance of success and shortens the induction–delivery interval The main argument against artificial rupture of the membranes is that it is an irrevocable step—a Rubicon that, once crossed, commits the obstetrician to delivery Perhaps it would be wiser to question the necessity for the induction, rather than the necessity for this procedure, which certainly increases the chances of a successful induction The only real risk from amniotomy other than infection is the risk of cord prolapse, but provided proper precautions are taken, this risk is of the order of 0 5% (Alderman 1975)

Oxytocin

Oxytocin (Pitocin) infusion alone will succeed in inducing labor near term in only about 70% of patients (Spellacy *et al*, 1973) Even with two infusions, the failure rate is still 20% (Cunningham *et al*, 1976) The high failure rate has prompted some practitioners to repeat the infusion daily until labor eventually starts This approach often has a deleterious effect on patient morale, but provided the usual precautions are observed, it has no other ill effects Such an approach is, of course, not suitable for the patient in urgent need of delivery Other practitioners combine oxytocin infusion with artificial membrane rupture

Oxytocin is usually administered by the intravenous route as a dilute solution, 10 units of oxytocin in 1000 ml of a carrier solution, usually 5% dextrose in water Details of dosage and administration of oxytocin are similar for both induction and stimulation of labor and have been described in Chapter 9 The most important complication of oxytocin is the risk of producing uterine hypertonia, which may result in fetal distress or, if neglected, may even culminate in uterine rupture Fetal distress may occur in any patient, and its appearance mandates discontinuation of the infusion Dangerous hypertonia and even rupture can be produced in multiparous patients, even at very low doses of the

drug Further, the sudden onset of tumultuous contractions may cause amniotic fluid embolus All these dangerous effects can and do occur, and frequent usage of oxytocin without disaster is no reason to become complacent Careful monitoring of mother, infant, and infusion rate are absolutely essential

Prostaglandins

Prostaglandins of the E and F groups can be used successfully to induce labor They may be administered as a dilute intravenous infusion or in tablet form

INTRAVENOUS PROSTAGLANDINS Intravenous prostaglandins E_2 and $F_{2\alpha}$ are no more successful in inducing labor than oxytocin, whether accompanied by amniotomy (Brown *et al*, 1973) or not (Spellacy *et al*, 1973) Early suggestions that they would prove to be devoid of the hazard of uterine hypertonia have not been borne out by experience Hypertonia does indeed occur, and uterine rupture has been described (Brudnell and Chakravarti, 1975) Additional side effects include phlebitis and nausea and vomiting However, intravenous prostaglandins may be considered in cases of intrauterine fetal demise, as they appear to have some advantages in this situation (Gordon and Pipe 1974)

ORAL PROSTAGLANDINS Oral prostaglandin E_2 has been extensively tested as an agent for induction of labor or for 'priming' the uterus prior to induction by amniotomy and oxytocin Many encouraging reports have appeared (Lauersen and Wilson, 1974, Miller *et al*, 1975, Cunningham *et al*, 1976), but nausea, vomiting, and transient fever have been described, as has severe uterine hypertonia (Felmingham *et al*, 1976) Oral prostaglandin E_2 is a promising drug, but its place in the obstetric armamentarium has yet to be determined

Intraamniotic Corticosteroids

Mati *et al* (1973) successfully induced labor in a small group of postmature patients by injecting 20 mg of betasone into the amniotic cavity Similar results were obtained by Nwosu *et al* (1976) and Craft *et al* (1976), but very small numbers of patients were involved in these studies, and the technique must, at this stage, be considered experimental

VERSION

Version is a procedure designed to alter the polarity of the fetus in the uterus Three types are usually described—external, internal, and bipolar The latter two are almost never used nowadays in singleton pregnancies A description of internal version of the second twin is included in Chapter 9 Before any type of version is carried out, an underlying cause for the malpresentation should be sought

EXTERNAL VERSION

Indications

1. Conversion of a breech to a cephalic presentation
2. Conversion of a transverse lie to a cephalic presentation

Criteria for Safety and Success

1. The abdominal wall and uterus must be relaxed The procedure should never be attempted during a contraction
2. The membranes must be intact and there must be sufficient fluid in the amniotic sac
3. The maternal bowel and bladder should be emptied
4. The presenting part is dislodged from the lower uterine segment and held above the pelvic brim before version is commenced
5. Excessive force must never be used

Dangers

1. *Premature labor*
2. Cord prolapse
3. Placental separation

Technique for External Cephalic Version

Before external version is considered, all obstacles to vaginal delivery must be ruled out

1. The patient should be placed in a slight Trendelenburg position on a hard couch, with the bladder and bowel empty The obstetrician should stand to the side of the patient
2. Dusting powder is applied to the abdomen so that the obstetrician's hands may slide over the skin surface more readily

RIGHT SACROANTERIOR POSITION When the baby is in the right sacroanterior position, the breech is dislodged from the lower uterine segment and drawn upward toward the left side of the mother's abdomen with the lower hand At the same time the obstetrician's other hand intermittently and jerkily presses the head downward and toward the right lower quadrant while the head is kept as flexed as possible so that the fetus performs a backward somersault (Fig 10-1A) When the fetus reaches the transverse lie situation, the fetal heart should be checked, and if this is less than 80 beats per minute, no further version should be carried out If the fetal heart rate does not return to normal within a minute, the possibility that cord traction is being exerted should be considered and attempts at version discontinued So far as is possible, the fetus should be allowed to turn of its own accord

Alternatively, or if this method fails, an attempt should be made to rotate the fetus in a forward somersault maneuver (Fig 10-1B) This latter method has the disadvantage of bringing the umbilical cord over the cervix and increasing the danger of presentation or prolapse of the cord It carries the advantages, however, of facilitating version by improving fetal flexion and reducing cord traction

The patient should be observed for 30 min after attempted version for any evidence of bleeding or fetal distress If easy external version cannot be performed, the procedure should be abandoned No anesthesia should be given to facilitate external version for under these circumstances too much force is liable to be applied by the obstetrician

The ideal time for external version is about the 34th week of pregnancy Be-

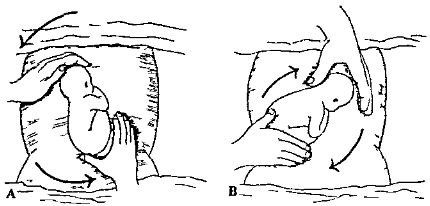


FIG 10-1 External cephalic version A Breech in right sacroanterior position is dislodged from lower uterine segment and drawn upward toward left side of mother s abdomen with obstetrician s right hand Simultaneously, the left hand jerkily presses head downward and toward right lower quadrant causing fetus to perform a backward somersault B External cephalic version by forward somersault method Head is kept well flexed to facilitate version Fetal heart should be checked when baby is in transverse position (half way) So far as possible baby should be allowed to complete the version by its own limb movements Excessive force must never be used

fore this time most breeches will turn spontaneously, and after this time version will become difficult About 75% of the breech presentations can be turned at the 34th week Although undoubtedly vertex delivery carries a lower fetal perinatal mortality than breech delivery, it should be borne in mind that the mortality from external version alone under anesthesia is about 2%

TRANSVERSE LIE When a transverse lie is present, external cephalic version should be attempted just before, or soon after, the onset of labor, *provided the membranes are intact*

LEFT SCAPULOANTERIOR POSITION In the case of a patient with a fetus in the left scapuloanterior position the routine is similar to that for version of a breech

1. The patient is placed in the supine position and the obstetrician stands at her side
2. Dusting powder is applied to the abdomen
3. The breech is grasped and drawn upward with the lower hand while the other hand directs the head over the pelvic brim The head must be kept well flexed
4. Fundal pressure is applied so that the vertex is pushed downward into the pelvis

FORCEPS DELIVERY

The obstetric forceps is an instrument designed to accelerate delivery of the infant The instrument consists of two interlocking parts whose blades, when assembled, form a cradle (cephalic curve) which surrounds the fetal head

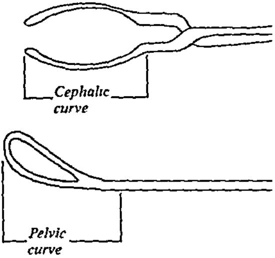


FIG 10-2 The cephalic curve of the forceps is designed to encompass fetal head pelvic curve is designed to fit pelvic canal

Below the blades are the shanks, lock, and handles The blades and handle are at an angle (pelvic curve) unless the forceps are designed for a special purpose (Fig 10 2) *The forceps, properly used, is an indispensable instrument, and it is used in as many as 30% of vaginal deliveries* (Barden 1975)

INDICATIONS FOR FORCEPS DELIVERY

Prolongation of the Second Stage of Labor

There appears to be little doubt that the longer the second stage of labor continues, the more likely it is that the infant will become hypoxic The old rule was that the second stage of labor should be terminated after 2 hours in the primipara and after 1 hour in the multipara This arbitrary time limit is often shortened by as much as half in modern obstetrics, indeed many obstetricians intervene *once progress ceases Intervention should be considered if the progress of labor stops,* but the best interests of each mother and infant should be carefully weighed before the obstetrician embarks upon an operative delivery Although a low forceps 'lift out' may prevent the development of hypoxia in a fetus whose progress is arrested by a rigid perineum a midforceps rotation and extraction may even cause fetal damage, the very thing it is intended to prevent Thus, *an easy outlet forceps procedure is desirable, but passage of a prescribed length of time may not be a strong enough indication for a mid forceps* operation A potentially difficult mid cavity forceps delivery often becomes an easy low forceps delivery with the passage of 15 min, and provided the fetal heart rate is carefully monitored, such delay is permissible A policy of active management of labor will greatly decrease the number of potentially dangerous forceps deliveries

Fetal Distress

Evidence *of fetal distress in the second stage of labor should be managed by prompt delivery of the infant* The mode of delivery selected will depend on the station and position of the fetal head Prophylactic low forceps delivery

should be considered for fetuses in whom there is prior evidence of growth retardation or placental insufficiency

Maternal Distress

Any patient to whom the strain of the second stage of labor is truly deleterious should be delivered expeditiously Patients suffering toxemia, chronic hypertension, cardiac or respiratory disease, or other conditions in which strenuous exertion is dangerous should be delivered by forceps as soon as it is safe to do so

ESSENTIALS FOR SUCCESSFUL FORCEPS DELIVERY IN A VERTEX PRESENTATION

1. The fetal head must be presenting and engaged in the pelvis, and the exact position known
2. The membranes must be ruptured
3. The cervix must be fully dilated and retracted
4. The bladder and bowel must be empty Catheterization is probably not necessary for a low forceps operation, but should be done prior to midforceps application
5. There must be no obstruction at or below the fetal head This includes disproportion, pelvic tumors, etc.
6. The forceps must be applied cephalically—*i e*, each blade should come to lie over the appropriate parietal bone

These conditions are founded on experience gained over many years by many obstetricians, anyone who ignores them does so at their own and their patients' peril

TYPES OF FORCEPS DELIVERY

Outlet Forceps

When the fetal head is in the occipitoanterior position on the pelvic floor and visible at the introitus between contractions application is described as an outlet forceps delivery This is usually an elective procedure

Low Forceps

The same as an outlet forceps except that some rotation is required This is usually an indicated procedure

Midforceps

When the fetal head is engaged in the pelvis but has not descended to the pelvic floor, the operation is described as a midforceps delivery

Rotation Forceps

A mid-cavity forceps delivery in which the fetal head is rotated to the occipitoanterior position prior to applying traction is termed rotation forceps delivery

High Forceps

This term describes the application of forceps prior to engagement of the fetal head There is no place for this procedure in modern obstetrics

TECHNIQUE OF FORCEPS DELIVERY

The technique of forceps delivery is described in all standard textbooks of operative obstetrics, so a detailed description of technique will not be included here, but the procedure is illustrated in Figures 10-3 and 10-4

Assessment

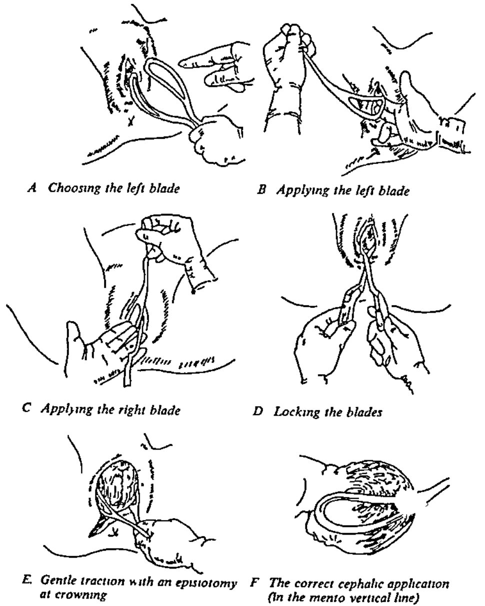
Before an operative delivery is undertaken it is imperative to assess the situation carefully Other methods of management should be at least considered before attempting a midforceps extraction Alternative methods of management include watchful waiting, delay, oxytocin infusion vacuum extraction, and cesarean section and, in any individual case, one of these may be more appropriate to the situation Engagement of the fetal head is said to occur when the biparietal diameter has passed through the pelvic inlet The most dependent part of the fetal head will usually be at or just below the ischial spines It must be remembered, however, that when there is excessive caput or molding, the biparietal diameter may lie *higher* than expected, and forceps delivery may not be the appropriate management

Application

If difficulty is encountered with application of the forceps it is likely that 1) there is an unsuspected malpresentation (brow or mentoposterior face), 2) the cervix is not fully dilated, 3) the head is not engaged or 4) the position has been incorrectly diagnosed In all except the last case, further attempts at forceps delivery should be abandoned In the case of incorrect diagnosis of the position of the fetal head, reapplication of the blades in the correct cephalic application will solve the problem The same considerations apply to difficulty with locking the handles, if they do not come together easily the situation should be reassessed The handles should never be forced to lock

Traction

The most important point in applying traction is the correct positioning of the obstetrician operator When the patient is in the lithotomy position the direction of the birth canal—and therefore the direction in which the fetus progresses—is caudal and backwards until it comes to the pelvic floor (Fig 10-5) The obstetrician should therefore be positioned on a low stool with his hands below the vulva, so that he is able to exert traction in these directions When the vertex is in the occipitoanterior position, traction should be exerted in a downward and backward direction When the vertex is in the occipitoposterior position the direction of traction is more horizontal Traction should be applied with only one hand, should be intermittent and should coincide with uterine contractions if possible The axis traction attachment is very useful for ensuring



A Choosing the left blade *B Applying the left blade*

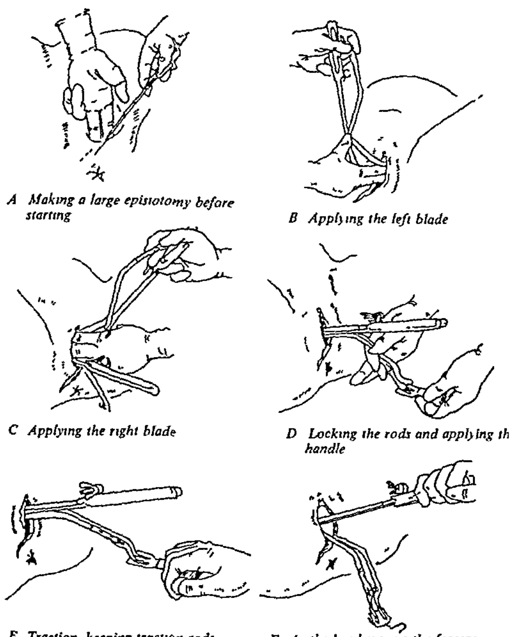
C Applying the right blade *D Locking the blades*

E Gentle traction with an episiotomy at crowning *F The correct cephalic application (in the mento vertical line)*

FIG 10-3 Low forceps delivery (Obstetrics Illustrated London, E&S Livingstone Ltd 1969)

that traction is in the right direction, but care must be taken not to use excesssive force with it Once the fetal head has descended onto the pelvic floor some gentle backward traction should be maintained, as this helps to maintain the attitude of flexion and prevent extension of the episiotomy

If gentle, one handed traction does not result in descent of the head, the situation should be reassessed The usual cause of this will be one of the points cited in the foregoing section (Application), but if no cause is apparent,



A Making a large episiotomy before starting

B Applying the left blade

C Applying the right blade

D Locking the rods and applying th handle

E Traction, keeping traction rods parallel to the shank

F As the head crowns the forceps are held by handles and the head is lifted over the perineum

FIG 10-4 Midforceps delivery (Obstetrics Illustrated London E&S Livingstone Ltd 1969)

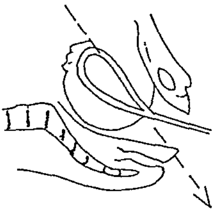


FIG 10-5 Line of traction for midcavity forceps delivery

it is reasonable to presume that cephalopelvic disproportion is the likely cause and to deliver the patient by cesarean section Under no circumstances should very strong traction be employed

VACUUM EXTRACTOR (VENTOUSE)

This instrument has become popular and in many parts of the world has largely replaced the forceps The idea of assisting delivery by applying a vacuum to the head of the fetus dates back to the early 18th century, but it was not until Malmstrom (1953, 1957) developed his instrument that its use became widespread Many modifications of his instrument are now available, but each consists of three basic components suction cups, a vacuum pump, and a traction device

EQUIPMENT

Suction Cups

There are usually three cups with different diameters The design of the cup is such that when the vacuum is built up, the scalp fills the dome of the cup, which is of larger diameter than the mouth of the cup (Fig 10-6), thus allowing traction to be used without detaching the cup The "knob" on the cup is pointed toward the occiput to enable the operator to observe rotation

Traction Device

The original Malmstrom system (Fig 10-7) for creating the vacuum is quite adequate, provided the operator remembers to use the traction bar and not to pull on the tubing The Bird modification (1969) placed the vacuum tube in the side of the knob while leaving the traction chain at the center, thus avoiding the problem of accidental detachment of the tubing This has been further modified (Fig. 10–8)

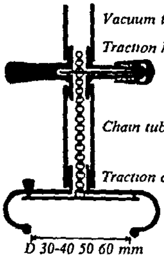


FIG 10-6 Diagrammatic section of Malmstrom's vacuum extractor Malmstrom T Acta Obstet Gynecol Scand 36 (Suppl 3) 5, 1957

Vacuum Pump

The simple hand pump with suction bottle and attached vacuum gauge (calibrated in kilograms per square centimeter), designed by Malmstrom is still largely in use The pump must be handled by an assistant, and various designers have produced foot-operated and hand operated pumps, as well as electric pumps, which may be used by the operator The simple vacuum system shown in Figure 10 7 is, however, perfectly adequate

INDICATIONS FOR VACUUM EXTRACTION

The vacuum extractor may be used in almost any circumstance in which forceps delivery is indicated However, it should not be used in a face presentation because of the danger of soft tissue damage, nor should it be used in acute fetal distress in the second stage of labor, when a forceps delivery can be completed much more rapidly

In addition to the usual indications for forceps application, the vacuum extractor may be applied before full dilatation of the cervix, as in patients with flagging labor or severe cardiopulmonary disease The instrument should never be used unless the presenting head (not caput) has reached zero station Both Lange (1961) and Fjallbrant (1964) have demonstrated that high vacuum extraction is as obsolete as the high forceps operation The only exception to this is its use with a second twin

TECHNIQUE

The patient is placed in the lithotomy position General anesthesia is not only unnecessary but undesirable, as the patient's cooperation is required Epidural or pudendal block is adequate The largest cup that the dilation of the cervix will allow is selected The instrument should be assembled and the vacuum tested against the gloved hand If there are no leaks, the cup is then placed against the fetal head as near the occiput as possible

A finger is swept around the cup to ensure that no cervical or vaginal tissue is trapped inside the cup Next, a vacuum of $-0\,2$ kg/cm² is induced, and another check for interposition of maternal tissue is made Thereafter, the

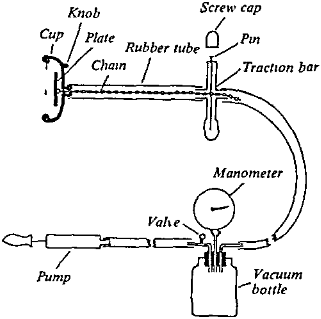
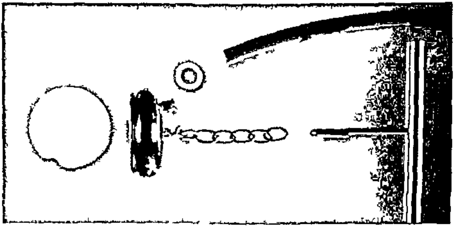


FIG 10-7 The Malmstrom apparatus for creating vacuum

FIG 10-8 B rd s modification of the cup of the Malmstrom vacuum extractor wi traction chain and handle (Courtesy of AB Vacuum Extractor Gothenburg Sweden) Vacuum correction and tract on chain have be separated



vacuum is increased by 0 2 kg/cm² every 2 min until an effective vacuum of 0 8 kg/cm² is attained This vacuum may be induced more rapidly if delivery is urgent, but the steps of 0 2 kg/cm² every 2 min allow for maximum formation of artificial caput (chignon) inside the cup, and more rapid induction of vacuum often results in detachment of the cup when traction is applied

Traction should always be at right angles to the cup, and should be applied synchronously with uterine contractions If traction is not applied at right angles to the cup, the shearing force applied may be sufficient to cause detachment of the cup If the head is in the midcavity of the pelvis an episiotomy may be necessary to ensure the correct angle of traction Traction is applied with one hand only, the fingers of the other hand being placed against the cup to ensure that the correct angle of traction is maintained

ADVANTAGES AND DISADVANTAGES OF THE VACUUM EXTRACTOR

The main advantage of this instrument is that it may safely be applied before full cervical dilatation It is also very difficult to use excessive force, as this will result in detachment of the cup rather than maternal or fetal injury Traction forces of more than 15 kg will result in detachment This is much less force than can be obtained, sometimes unwittingly with forceps (Donald, 1969, Chalmers, 1975) In cases of occipitoposterior or occipitotransverse positions, the fetal head is allowed to rotate at whatever level it finds easiest Chalmers (1968) found that 30% of occipitoposterior positions delivered by vacuum extraction did not rotate to occipitoanterior Rotation is never forced The absence of forced rotation decreases the incidence of vaginal lacerations, because the diameter of the presenting part is not increased as with the forceps

The main disadvantage is the length of time (6–8 min) it takes to obtain the optimum pressure Many complications such as scalp detachment, annular detachment of the cervix, and subgaleal hematomas were attributed to this instrument when it first came into vogue Provided the following precautions are observed, these problems should not arise

1. The cup should never be left on the fetal head for more than 30 min If delivery has not occurred by this time, further efforts are unlikely to be successful (Chalmers 1975), and prolonged application of the vacuum may result in scalp necrosis
2. The vacuum should never be used when the fetal head is above the pelvic brim
3. When the cup has been positioned on the fetal head, a careful check should be made for inadvertent inclusion of maternal tissues
4. Traction should be applied only during contractions and should always be at right angles to the cup

CESAREAN SECTION

INDICATIONS

It is customary to list the indications for cesarean sections as maternal or fetal but as the interest of mother and fetus may coincide (fetopelvic disproportion) or conflict (prolonged rupture of membranes with fetal distress) they are here listed without this division

1. Cephalopelvic disproportion Cesarean section is used in this situation after adequate trial of labor in vertex presentations
2. Previous cesarean section When the indication is still present, repeat cesarean section is indicated—e g, fetopelvic disproportion In the United States the practice of "once a section always a section" is usually followed However, when the indication for the cesarean section is no longer present, a trial of labor may be considered Myomectomy scars that extend into the uterine cavity and hysterotomy scars are sometimes indications for cesarean section
3. Acute fetal distress Cesarean section is indicated when rapid atraumatic vaginal delivery is not feasible Among such situations, of course, are prolapsed cord, and abruptio placentae
4. Chronic fetal distress Signs of fetoplacental deterioration when conditions are not favorable for induction of labor are indications for cesarean section This applies particularly to patients with diabetes mellitus, rhesus isoimmunization, the elderly primigravida, and the patient with a poor obstetric history
5. Failed induction If there is an indication for induction of labor, the physician should be prepared to deliver the patient by cesarean section, should the induction fail
6. Placenta previa All but very minor grades of placenta previa should be delivered by cesarean section
7. Pelvic tumors If a pelvic tumor obstructs delivery, whether the tumor is of uterine or ovarian origin, delivery by cesarean section is indicated
8. Malpresentations For details see Chapter 9
9. Intrauterine infection See Chapter 4
10. Previous gynecologic surgery A history of repair of either vesicovaginal fistula or stress incontinence is indication for cesarean section
11. Vaginal septums If these cannot be stretched or resected, cesarean section should be performed
12. Carcinoma of the cervix discovered in late pregnancy
13 Herpetic vulvovaginitis This is an indication for cesarean section, unless the baby is dead or the membranes have been ruptured for 4 hours or longer

TYPES OF CESAREAN SECTION

The types of cesarean section currently in use are presented in Table 10-2 Each is described in detail in standard textbooks of operative obstetrics, so a detailed description will not be included here

CHOICE OF ANESTHETIC

General anesthesia is always hazardous, particularly when the patient is or has been in labor, and/or has eaten recently Aspiration of acid stomach contents may occur in spite of nasogastric aspiration and skillful, rapid endotracheal intubation Administration of antacids every 2 hours during labor will prevent, or minimize, the effects of such aspiration (Crawford, 1972) Antacids should always be given if cesarean section is to be performed under general anesthesia

Both spinal and epidural anesthesia are adequate and, indeed, very satisfac-

TABLE 10-2. Types of Cesarean Section

Operation	Procedure	Advantages	Disadvantages
Classic (Upper segment)	Longitudinal incision in upper segment of uterus through contractile part of uterine wall	Rapid, suitable for transverse lie and some placentae previae	Hemorrhage, poor healing, bowel adhesions, poor involution, infection, may rupture before labor in subsequent pregnancy or during labor (6 4%)
Lower Segment Transverse	Transverse incision in lower segment, parallel to muscle fibers, scar extraperitoneal	Good healing, less infection and hemorrhage Incidence of subsequent rupture during labor low (1 4%) operation of choice	Takes longer; not suitable for transverse lie
Vertical	Same as for transverse except incision is through muscle fibers	None	Often extends into upper segment with problems as in classic type; may extend downward with risk of bladder injury
Extraperitoneal	Lower segment approached by dissecting peritoneum off bladder	Localizes infection extraperitoneally, recommended by some for infected cases	Technically difficult, hemorrhage and damage to bladder common

tory Local infiltration alone has been used successfully but is recommended only in cases of dire emergency when anesthesia is unavailable Indeed, it will often take longer to infiltrate the various layers than to seek out an anesthetist

POSITION

The importance of the supine hypotensive syndrome is stressed in Chapter 3 and 9 When the loss of vasomotor tone associated with anesthesia is added to the effects of caval compression, the effective blood flow in the uteroplacental circulation may be greatly decreased and the fetus severely depressed To obviate this the patient should be positioned on the operating table with a 15° wedge under her right side Infants delivered by cesarean section performed in the tilted position have a lower incidence of poor Apgar scores (Crawford, Barton, Davies 1972)

CHOICE OF INCISION

A longitudinal midline, or paramedian incision or a transverse Pfannenstiel incision can be used The transverse incision is more tedious but has many advantages over the midline incision Wound dehiscence is very rare with a transverse incision, and the incision is less painful a factor that encourages coughing and ambulation Exposure is also perfectly adequate The only disadvantage, other than the slight increase in operating time, is that on occasions repeat cesarean section may be quite difficult A Pfannenstiel incision should not be used for an emergency cesarean section or in any situation where a low vertical or classic section may be required

COMPLICATIONS

Hemorrhage is the most common operative complication *Brandt (1966) has shown that the average blood loss during cesarean section is about 1000 ml so blood should always be available for rapid transfusion*

Postoperative febrile morbidity can be expected in 33% of cases (Jones, 1976) Apart from endometritis, urinary tract and respiratory infections are common Serious pelvic infections with or without secondary hemorrhage may develop (Fig 10-9) and their management has been discussed in Chapter 4 Wound infections are more common in emergency cases, as are wound dehiscences requiring resuture Thromboembolic disease is 12 times more likely to occur following cesarean section than vaginal delivery The prevention early diagnosis, *and treatment of this potentially fatal complication* have been discussed in detail (Ch 4)

Although cesarean section has become a very safe operation in terms of maternal mortality, the mortality is still much higher than that expected from vaginal delivery More and more cesarean sections are being done strictly for fetal indications, often in circumstances in which the mother would be much better served by a vaginal delivery (amnionitis, prolonged rupture of membranes), so we can expect some increase in maternal morbidity The case for prophylactic antibiotics in cesarean section is unproved The general consensus appears to be that they are not indicated

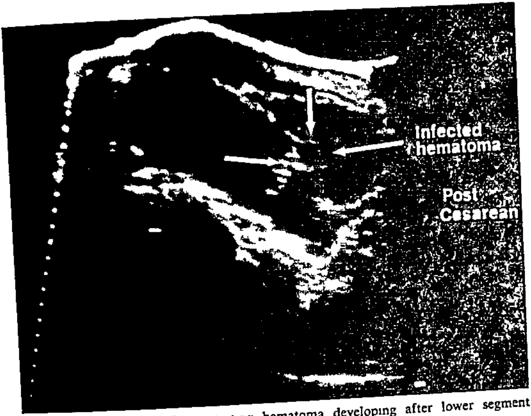


FIG 10-9 Sonogram demonstrating hematoma developing after lower segment cesarean section (Courtesy Dr R E Woods)

VAGINAL DELIVERY FOLLOWING CESAREAN SECTION

The dictum of "once a section always a section has general support However, the maternal morbidity following repeat cesarean section is 5–10 times higher than that following vaginal delivery In addition the incidence of scar rupture following lower segment cesarean section is low (1–3%), and if rupture of a low transverse scar should occur, the results are not as disastrous as if there is rupture of a classic scar These considerations have lead some obstetricians to allow trial of labor following cesarean section This is permissible provided the following conditions are fulfilled

1. The patient has had only one previous cesarean section
2. The indication for the previous operation is no longer present
3. The scar is of the low transverse variety (The incidence of rupture following the classic operation is approximately 6 4% [O'Driscoll 1966])
4. Facilities are available for immediate laparotomy
5. Two units of compatible blood are available
6. The patient is carefully monitored throughout labor (Very often the first sign of impending rupture is fetal distress, sudden cessation of contractions, and tenderness over the scar Sudden tachycardia may also be noted)
7. Any delay in labor is managed by prompt laparotomy (Oxytocin should never be used)
8. The second stage of labor is shortened if necessary

. The integrity of the scar is checked immediately after delivery by manual exploration of the uterine cavity

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Chapter 11

Postpartum Emergencies

Timothy C. F. O'Connor, Denis Cavanagh

Behold how great a matter a little fire kindleth!
The Epistle of St James III 5

Postpartum hemorrhage is defined as blood loss in excess of 500 ml following delivery It is called primary postpartum hemorrhage when it occurs within 24 hours of delivery, and secondary when it occurs after this time

PRIMARY POSTPARTUM HEMORRHAGE

This type of hemorrhage is usually described as atonic or traumatic It is, however, possible and even common for both forms to occur in the same patient

ATONIC POSTPARTUM HEMORRHAGE

Prevention

1. The incidence of postpartum hemorrhage may be reduced and its effects will certainly be less serious if anemia is recognized and treated in the antepartum period
2. Certain patients, by virtue of previous history, medical conditions, or complications of pregnancy, labor, or delivery are particularly prone to hemorrhage (see Table 11-1) Such patients should have two units of blood typed and cross matched during labor, oxytocin or ergonovine (Methergine), or both, should be given with the delivery of the anterior shoulder, and an anesthetist should be on hand until the third stage of labor has been completed
3. An intravenous infusion should be started in all laboring patients but this is especially important when postpartum hemorrhage is likely
4. The infant should always be delivered slowly
5. The third stage of labor should be conducted in the proper manner Improper conduct of this stage of labor contributes to excess blood loss The traditional method of waiting for signs of separation of the placenta (lengthening of the cord, change in size and shape of the uterus, and a gush of blood)

TABLE 11-1 Conditions that Predispose to Postpartum Hemorrhage

Conditions	
Antedating pregnancy	**Arising during pregnancy and labor**
Previous postpartum hemorrhage	Placenta previa
Grand multiparity	Abruptio placentae
Fibroids	Multiple pregnancy
Idiopathic thrombocytopenia purpura	Polyhydramnios
Von Willebrand's disease	Precipitate labor
Leukemia	Prolonged labor
	Chorioamnionitis
	Forceps delivery
	Cesarean section
	General anesthesia
	Mismanagement of third stage of labor
	Acute coagulation defect

without giving any oxytocics has, in many centers, been replaced by a more active management Two acceptable methods are the Pastore and the modified Brandt-Andrews maneuver The essential feature of these methods is that no manipulation is done until the uterus is firmly contracted Oxytocin is usually given prior to delivery of the placenta by either of these methods

MODIFIED BRANDT-ANDREWS METHOD (Fig 11-1)

A. Clamp the umbilical cord close to the vulva immediately after delivery of the infant
B. Place the left hand on the uterine fundus but do *not* massage it
C. When the uterus becomes firmly contracted, place the fingertips of the left hand between the symphysis and the fundus With the other hand, grasp the cord clamp firmly
D. Elevate the fundus with the left hand while applying gentle traction on the cord in a downward and backward direction to deliver the placenta

   While using controlled cord traction to deliver the placenta, the most important thing to remember is not to use traction until the uterus is well contracted, or acute inversion of the uterus may result (see Ch 3, Shock)

6. The cervix and vagina should be inspected for lacerations after every delivery In addition, the uterine cavity should be manually explored after all operative deliveries other than *outlet forceps, and also when there is any doubt about* the integrity of the placenta, without *waiting for the patient to start bleeding*
7. Even after delivery of the placenta the patient should be kept under careful observation for 2 hours in the delivery suite This period is so important that it has been named the fourth stage of labor

Management

Once excessive bleeding occurs, it is important to move quickly to minimize blood loss If the placenta is still *in utero* and not readily removable by one of the methods described above, manual removal should be done rapidly, preferably with the *patient under general anesthesia*

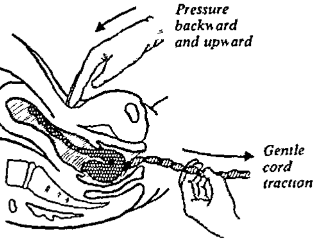


FIG 11-1 Brandt Andrews maneuver Pressure is applied at junction of upper and lower uterine segments with the fingers of the right hand in a backward and slightly upward direction Meanwhile, gentle cord traction with the left hand results in delivery of placenta (Redrawn from Greenhill JP Obstetrics 12th ed Philadelphia WB Saunders 1960)

TECHNIQUE OF MANUAL REMOVAL OF THE PLACENTA

A. The cord is held taut with the left hand while the right hand follows it up through the cervical canal to its insertion on the fetal surface of the placenta
B. The left hand is now placed on the abdomen grasping the fundus of the uterus through the abdominal wall The uterus is pushed downward and its stability maintained while the fingers of the right hand seek the place of separation at the lower margin of the placenta
C. The placenta is now separated through the spongy layer of decidua by a sawing motion of the right hand (Fig 11-2)
D. When the placenta is completely separated, it is slowly withdrawn, after awaiting contraction of the uterus over the exploring hand
E. Every time a placenta is delivered, whether spontaneously or manually, it should be carefully inspected to ensure that it is complete and that a succenturiate lobe has not been retained in the uterus Unfortunately, evidence of the latter is easy to miss and the lobe may only be found on very careful uterine exploration
F. As previously mentioned, the vagina and cervix should be carefully inspected for lacerations after the delivery of the placenta Re inspection is indicated when bleeding persists in the presence of a firm uterus

If bleeding persists despite oxytocin administration the uterus is elevated by seizing the corpus through the abdominal wall and compressing it against the vertebral column The left hand compresses the fundus while the lower uterine segment and cervix are compressed between the encircling thumb and fingers of the right hand (Fig 11-3) Apart from the effect achieved by compression of the uterus, the uterine veins are straightened by elevation reducing congestion of the uterine sinuses This method is not satisfactory in the obese patient If the uterus enlarges with clots despite compression, these should be expressed and the uterus regrasped and held upward as previously described.

If bleeding persists despite the measures described, bimanual compression

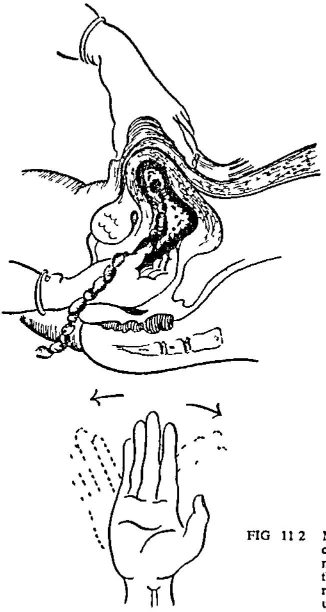


FIG 11 2 Manual removal of placenta A sideways sawing motion of the hand from the wrist is used to separate the placenta from the uterine wall

of the uterus should be tried (Fig 11-4) If after 5 min of compression a tendency to hypotonicity is still present, the posterior wall of the uterus should be massaged by the abdominal hand while the fist in the anterior fornix massages the anterior uterine wall by a gentle pronation supination movement So long as adequate compression is maintained and adequate blood replacement is carried out, the patient is safe If hypotonia persists after 30 min of compression combined with oxytocics, further steps must be considered to halt bleeding

Packing the uterus is a procedure that is rarely used in modern obstetrics

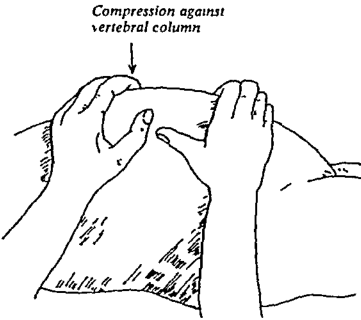
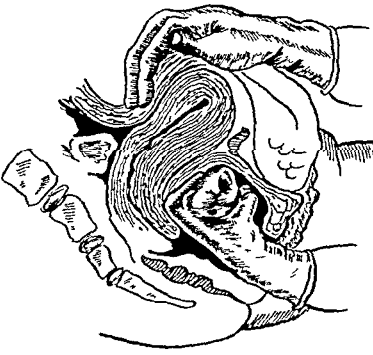


FIG 11-3 Dickinson's method of controlling postpartum hemorrhage The uterus is elevated with the right hand Compression of the uterine arteries is effected by the fingers of the right hand encircling the cervix while the corpus is compressed against the vertebral column

FIG 11-4 Bimanual compression of uterus Uterus is compressed between the clenched left fist in the anterior fornix and the right hand placed on the patient's abdomen.



Experience is required to pack a uterus properly, and the procedure itself is not without hazard, and it has been condemned by many obstetricians as unphysiological It has been claimed that it contributes to uterine atony, causes infection, and may delay diagnosis of uterine rupture Nonetheless, it may, on rare occasions, be a life-saving procedure (Hester, 1975) However, it may be successful only as a temporary measure

PROCEDURE FOR PACKING THE UTERUS Fifteen to 20 yards of dry, sterile gauze are used The left hand is positioned inside the uterus and the gauze fed to the fingers with a uterine packing forceps (Fig 11-5) The fingers of the left hand are used to pack the uterus firmly, starting at the cornua and working downward It is important to ensure that the fundus of the uterus is reached The vagina should also be packed The patient should be given antibiotics and a Foley catheter should be inserted The pack, if successful, should be removed after 12–24 hours

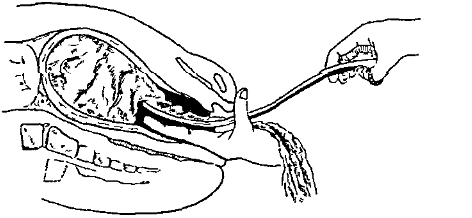
Because of the technical difficulty associated with uterine packing, various semi-automatic packing devices have been designed, but these are rarely available when needed (Torpin 1941)

Other methods of treating uterine atony unresponsive to oxytocics include the instillation of heated solutions into the uterus and the use of a gravity suit (Fribourg, 1973, Burdick, 1975)

If these measures fail to arrest significant hemorrhage and there is no evidence of trauma to the lower genital tract, laparotomy should be performed Hysterectomy may be the only way to control the hemorrhage, but the effect of internal iliac artery ligation may be tried first, if the patient is anxious to retain her childbearing potential (O'Leary and O Leary, 1966, Burchell, 1968)

Any patient who has persistent postpartum hemorrhage should have coagulation studies performed to exclude the presence of an underlying acute coagulation defect The investigation and treatment of this entity has been discussed in Chapter 2

FIG 11-5 Packing the uterus The left hand serves as a cervical speculum while the uterus is tightly packed with gauze, with the aid of uterine packing forceps The procedure is rarely necessary



'RETAINED PLACENTA' WITHOUT BLEEDING

Uterine Atony

Failure of the uterus to contract well enough to detach and expel the placenta is known as uterine inertia It is usually accompanied by hemorrhage sooner or later The effect of oxytocics may be tried, but if these fail, manual removal of the placenta should be performed The longer the placenta remains in the uterus, the more likely the patient is to bleed Most obstetricians accept 30 min delay as the criterion for intervention, and many would intervene before this Because manual removal tends to increase the total amount of third stage *bleeding in some patients, a delay of 30 minutes is reasonable*

Contraction Ring

This situation may occur following excessive handling of the uterus usually after the administration of oxytocics The lower half of the uterus contracts strongly, trapping the placenta above it Hemorrhage is not usually a feature of this condition *The contraction ring may relax spontaneously, and there is no emergency, provided the patient is not bleeding Alternately, inhalation of amyl nitrite may* be used to relax the ring. If manual removal of the placenta becomes necessary, deep general anesthesia will usually be effective

Morbid Adherence of the Placenta

Although this is an intrapartum complication it is best dealt with here because *of its implication for postpartum hemorrhage Morbid adherence of the placenta* is a rare occurrence Some degree of adherence occurs once in every 2000 deliveries Three types are recognized

1. Placenta accreta The placenta is pathologically adherent to the myometrium because of paucity or absence of underlying decidua
2. Placenta increta The placenta invades the uterine muscle
3. Placenta percreta The invasion reaches the serosa of the uterus, and may cause uterine rupture

Previous injury to the endometrium caused by trauma infection, or surgery, and placenta previa are the main etiologic factors In a case of complete placenta accreta, there is no bleeding in the third stage of labor until an attempt is made to remove the placenta Partial placenta accreta causes early *hemorrhage Indeed, hemorrhage may be torrential The presence of placenta* accreta is revealed when an attempt is made at manual removal of a retained placenta thought simply to be the result of uterine atony Because vigorous attempts to separate the placenta may result in extreme hemorrhage or in *uterine perforation, complete placental removal should only be accomplished if it can be done gently Otherwise, hysterectomy is the only safe treatment*

TRAUMATIC POSTPARTUM HEMORRHAGE

If bleeding persists in spite of a well-contracted uterus the cause is most likely to be traumatic There may be lacerations of the perineum, vagina, cervix, or uterine wall Pelvic hematomas should be borne in mind when the condition of the patient is worse than the amount of blood loss would seem to warrant

Perineal Lacerations

Four degrees of perineal laceration are generally recognized

1. A first-degree tear extends through the skin and superficial tissues to the muscle
2. A second-degree tear extends into the perineal muscles
3. A third-degree tear extends through the perineal muscles and the anal sphincter
4. A fourth-degree tear extends through the anal sphincter and involves the anterior wall of the anal canal or rectum

First- and second degree lacerations are not actual emergencies The more extensive tears will be discussed Considerable blood loss and possibly shock may result from a severe perineal laceration Blood loss in excess of 500 ml should be replaced and the laceration carefully repaired in layers

Even when blood loss is not excessive, lacerations involving the rectal sphincter require immediate and careful repair When these are neglected, fecal incontinence is a common sequel The belated repair of a complete tear or fecal fistula yields a success rate inferior to that with a carefully performed primary closure The following steps are required in repair of a fourth degree laceration

1. After the placenta is delivered, the vagina and perineum are washed thoroughly with antiseptic solution The patient is kept in the lithotomy position
2. The rectal and anal mucosa is closed with interrupted sutures of 3-0 chromic catgut. This is reinforced with a layer of interrupted Lembert sutures of 2-0 chromic catgut taken through the rectovaginal fascia and tied on the perineal aspect of the laceration
3. The retracted ends of the torn anal sphincter are situated at about the 10 and 2 o'clock positions They are grasped with Allis tissue forceps, pulled upward, and held together in the midline In this position they are approximated with interrupted 2-0 chromic catgut sutures taken through the muscle and its fascial sheath One retention suture of nylon may be taken through the thin subcutaneous tissues and external anal sphincter to provide further reinforcement but usually is not necessary
4. The levator ani muscles are approximated in the midline with a few 2-0 chromic catgut sutures
5. The deep and superficial transverse perineal muscles are approximated with interrupted 2 0 chromic catgut sutures
6. The vaginal mucosa is closed with a continous suture of 2-0 chromic catgut on an atraumatic needle Great care must be taken to ensure that the highest "bite" is taken above the apex of the vaginal laceration (If the laceration extends high in the vagina, it will be found more convenient to start the closure of the vaginal mucosa before approximating the perineal muscles)
7. The perineal skin and subcutaneous tissues are closed with a subcuticular suture of 3-0 chromic catgut

In the postpartum period a low-residue diet should be given for 7 days A stool softener such as dioctyl sodium sulfosuccinate (Colace) is given at night No enemas should be given

Vaginal Lacerations

Serious vaginal lacerations are more common in women on whom operative rotations and midforceps delivery have been performed The possibility is reduced by the timely performance of an adequate episiotomy

Bleeding points should be ligated and the lacerations closed with 2–0 chromic catgut sutures The repair of sulcus tears requires good exposure Lacerations in the region of the urethra and clitoris should be repaired with 3–0 chromic catgut Bleeding from vaginal varicosities is controlled with packing When bleeding is severe and the site of the laceration cannot be visualized adequately, the vagina should be tightly packed with a large gauze roll It should be appreciated that vaginal packing may enlarge the laceration, so the patient must be carefully observed for evidence of shock while the pack is in place After about 3 hours, the field will usually be dry and the laceration can easily be closed

Cervical Lacerations

Good lighting, an assistant, vaginal retractors, at least three sponge holding forceps, and an adequate supply of gauze swabs are all necessary for repair of a cervical laceration

The cervix should be pulled down using two sponge holding forceps These may be "leap-frogged" over one another all around the cervix to ensure that a laceration is not missed

Another useful method of detecting a cervical laceration when it cannot be visualized has been described by Graber and O Rourke After delivery of the placenta, the hand is introduced into the vagina, and the uterus is pushed upward into the pelvis The middle finger is introduced into the cervical canal for about 2 cm, and the index finger is placed over it on the vaginal aspect of the cervix so that the cervical tissue lies between them The fingers, maintained in this relationship to one another, are swept slowly around the cervix in a counter clockwise direction until the 6 o'clock position is reached The position of the fingers is now reversed, the index finger being inside the canal and the middle finger outside The counter-clockwise sweep is continued to the 12 o'clock position If a laceration of the cervix is present, the sweeping fingers will meet at the site of the defect (Fig 11 6) The most common sites for cervical lacerations following delivery are in the 3 o'clock and 9 o'clock positions They are relatively rare on the anterior lip of the cervix

It should be remembered that the purpose of suturing a cervical laceration is to stop hemorrhage Therefore, suturing a laceration that is not bleeding may be unwise because the cervix is often edematous and friable and the sutures easily cut through the tissues and may actually cause hemorrhage

The laceration should be sutured with interrupted 2-0 chromic catgut on an atraumatic round bodied needle The first suture should be placed above the apex of the laceration, but if visualization is difficult, it is often easier to place the first suture at the base Then with gentle traction on the first suture, guided by a finger inside the cervix, the remaining sutures may be placed in ascending fashion If difficulty is encountered because the sutures are cutting through the cervix, hemostasis may be often secured by placing sponge forceps on the lacerated edges

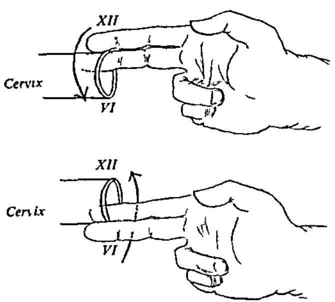


FIG 11 6 Detection of a cervical laceration by palpation The index and middle fingers meet at the site of a laceration

PELVIC HEMATOMAS

The development of a postpartum pelvic hematoma is not uncommon and occasionally may be of such magnitude as to result in hemorrhagic shock

Hematomas do not necessarily derive from lacerations of the birth canal or episiotomies They are usually the result of damage to a vessel wall without laceration of the overlying tissues This trauma may result 1) from pressure of the presenting part on the pelvic structures during the course of normal labor, 2) from forceps manipulation 3) from excessive external fundal pressure on the uterus, or 4) from paracervical or pudendal anesthesia

The apex of an episiotomy incision should be checked immediately after repair to ensure that a hematoma is not forming

All hematomas should be evacuated to avoid infection and subsequent abscess formation A dissecting hematoma can often be controlled by placing a 1 0 chromic catgut suture above the apex of the hematoma This suture should be placed deeply, with a finger in the patient s rectum to avoid injury to this structure An acute coagulopathy may be the causative factor in susceptible cases (see Ch 2 Clotting Disorders in Pregnancy)

Usually pelvic hematomas develop insidiously with the gradual extravasation of blood into the pelvic tissues The condition should always be suspected in patients who complain of increasing pelvic and perineal pain following delivery Often such pain is accompanied by sudden inability of the patient to void after she has done so easily earlier This is practically pathognomonic of pelvic or vulvar hematoma The diagnosis is readily made on vaginal or rectal examination In a recent study of the subject Pieri described three main sites of occurrence in relation to the fascial anatomy of the pelvis (Fig 11 7)

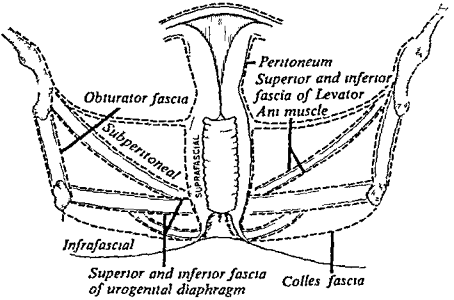


FIG 11 7 Types of pelvic fascia Pelvic hematomas may be classified as intra fascial, suprafascial and subperitoneal

Vulvar Hematoma

Following delivery, the patient complains of increasing perineal discomfort and pain At first no abnormality may be apparent, but later the area becomes tense and ecchymotic If the patient is in shock adequate blood replacement should be carried out

No time should be wasted in attempts at aspiration If the tense, swollen area appears to be associated with an episiotomy, the latter should be broken down and the hematoma 'evacuated' If there is no obvious laceration, the skin over the mass should be incised and the blood clots evacuated Any bleeding vessels that are visible should be ligated After bleeding has been controlled, all cavities should be obliterated, using deep mattress sutures of 1-0 chromic catgut on an atraumatic needle When hemostasis has been attained, the vaginal mucosa and vulvar skin are approximated with interrupted sutures of 2-0 chromic catgut In general, the application of a large gauze pressure dressing is to be preferred to gauze packing of a vulvar hematoma cavity A T-binder may be used to help maintain pressure

Because there is a tendency for reflex retention of urine to occur, it is usually advisable to insert a self retaining catheter for at least 24 hours

Vaginal Hematoma

The complaint of progressive pelvic pain should prompt vaginal and rectal examination On vaginal examination, the tender fluctuant mass indicating the presence of a hematoma is palpable below the mucosa

A longitudinal incision is made over the mass, and the hematoma evacuated Any obvious bleeding points should be ligated and the hematoma cavity lightly packed with oxidized cellulose (Oxycel) gauze Tight packing of the cavity

should be avoided because of the excessive friability of the walls A tight gauze pack should be inserted into the vagina to give adequate tamponade A Foley catheter should be inserted into the bladder Both the vaginal gauze pack and the Foley catheter are removed after 24 hours

Retroperitoneal Hematoma

Extension of a cervical laceration into the lower uterine segment may result in retroperitoneal hematoma, which may also occur in primiparas after a spontaneous labor Bleeding is usually due to the injury of the uterine or other branch of the anterior division of the hypogastric artery but may occur from an unligated vessel at the apex of the episiotomy

This condition should always be suspected in a patient who shows evidence of shock in the absence of significant external bleeding The diagnosis can be made on pelvic examination from the finding of a tender mass in the region of the broad ligament

Serial hematocrit estimations are useful in following patients in whom retroperitoneal bleeding is suspected

When evidence of hemorrhagic shock is present the patient should be adequately transfused and a laparotomy should be performed as soon as possible At operation, the hematoma may be found to extend upward to the level of the kidney The peritoneum should be incised and the hematoma evacuated If it is not possible to identify a definite bleeding point, the operator should proceed to ligate the anterior division of the hypogastric artery on the affected side The procedure is carried out also on the opposite side since the anastomosis between these two vessels is very great An attempt should be made to ligate the hypogastric artery below the level of the posterior division because sloughing of the buttocks has been reported in a few instances However, time should not be wasted to achieve this refinement in a patient in hemorrhagic shock

In patients with marked retroperitoneal extravasation, the anatomic structures are often difficult to identify, and great care must be taken to ensure that the ureter is not ligated In a few instances the extravasation may be from the ovarian vessels, if so, these should be ligated rather than the hypogastric arteries

SECONDARY POSTPARTUM HEMORRHAGE

Secondary postpartum hemorrhage is often due to retention of placental fragments or blood clot (Fig 11-8) It may also be caused by estrogen withdrawal when estrogens have been given "to suppress lactation" Frequently, it is associated with primary postpartum hemorrhage and febrile morbidity (Rome, 1975)

Bleeding may be heavy, and treatment consists of blood replacement and uterine curettage Great care must be taken not to perforate the uterus, which is often soft Perforation may occur in as many as 3% of cases, and if it should occur, laparotomy becomes necessary (Dewhurst, 1966) Oxytocin should be given prior to and during curettage to render the uterus firmer because of this danger, and because the placental fragments are often very ad-

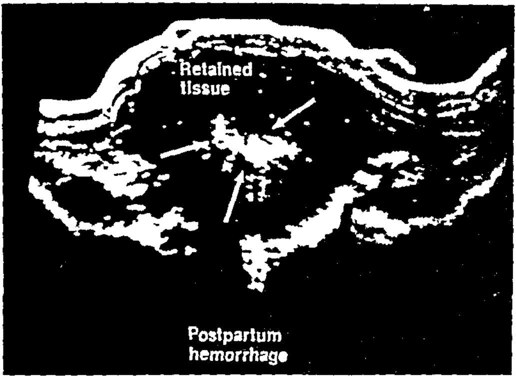


FIG 11 8 Postpartum hemorrhage Retained tissue demonstrated on sonogram This requires surgical evacuation

herent Curettage is usually effective even if no placental tissue is recovered Uterine packing or hysterectomy are only very rarely necessary

INJURIES TO THE URINARY TRACT

With the disappearance of heroic efforts to achieve vaginal delivery at whatever cost, serious bladder injury with or without fistula formation is becoming very rare Nonetheless the appearance of blood stained urine during labor or following delivery may give cause for concern Such cases are best treated by continuous bladder drainage until the urine has cleared

Vesicovaginal fistulas may be caused by pressure necrosis of the neck of the bladder as a result of prolonged labor or direct instrumental trauma In the former case, the fistula may take a week to become apparent, in the latter, it will be evident almost immediately In either case, the patient will complain of total incontinence of urine The diagnosis is confirmed by speculum or digital examination If necessary methylene blue dye may be instilled into the bladder to aid visualization

The patient should be managed by continuous gentle suction drainage of the bladder and encouraged to lie in the prone position Urinary antiseptics should be employed and frequent urinary cultures obtained With this regimen many fistulas close spontaneously Operative repair becomes necessary if spontaneous

closure does not occur by 3 months after delivery and should not generally be attempted before this time

INJURIES TO THE PELVIC GIRDLE

SYMPHYSIS PUBIS

The symphysis pubis may rupture partially or totally Rupture may be spontaneous or traumatic, and may occur before or during labor The condition is frequently associated with operative delivery

The patient may hear a crack or notice a bursting sensation at the time of rupture Pain over the affected joint, radiating down the thighs, is common, and difficulty with ambulation is present to a greater or lesser degree The diagnosis is made by feeling the wide gap between the pubic bones and is confirmed by x-ray films It is important to check that no damage to the sacroiliac joints has occurred Treatment consists of rest until the pain has subsided If pain is severe, local anesthesia may be injected around the joint If spontaneous healing does not occur, orthopedic surgery may become necessary

"Separation of the Symphyses" is a more common condition It usually occurs during the third trimester and its onset is gradual The main problem then becomes ambulation during the remainder of pregnancy This and the pain can be greatly alleviated by bed rest and the use of a broad, tight belt that snugly encircles the trochanters The latter is especially valuable for mild cases late in pregnancy, permitting comfortable ambulation Also, its use even while the patient is at bed rest increases comfort and hastens healing

COCCYX

The coccyx is bent backward at delivery, and if the sacrococcygeal joint is ankylosed, the coccyx may fracture The crack of the breaking bone is often clearly audible at delivery, and the diagnosis can be confirmed by eliciting crepitus and pain on movement of the coccyx The patient usually complains of tenesmus and has to support her weight on either trochanter when sitting Treatment consists of infiltration of local anesthesia, analgesics, local heat, and bed rest If spontaneous healing does not occur in 6 months, orthopedic surgery becomes necessary

MATERNAL OBSTETRIC PARALYSIS

This name is given to a rare lower limb paralysis that may occur during or just after labor It takes the form of "drop foot," and the incidence is between 1 2000 and 1 6000 (Cole 1946) There has been much debate in the past about the cause of this lesion, and three possible causes are cited

1. Damage to the Lumbosacral Trunk This large nerve trunk passes over the ala of the sacrum, and at the pelvic brim only weak fascia and peritoneum protect it from the fetal head Direct damage to the posterior fibers of this nerve trunk will produce a drop foot Many cases of drop foot occur following forceps delivery or following spontaneous delivery of a large infant

2. Lumbar Disc Protrusion O Connell (1958) pointed out that many cases of so-called drop foot also had weakness of the quadriceps and other muscle groups whose nerve supply is not derived from the lumbosacral trunk
3. Damage to Nerves Outside the Pelvis Pressure on the common peroneal *nerve as it winds around the neck of the fibula can produce a drop foot syndrome Such pressure may be produced by improper positioning of the patient in stirrups or leg holders*

CLINICAL PICTURE

Symptoms may present themselves in late labor, usually pain shooting down the affected leg, made worse by contractions More commonly, symptoms first appear the day following delivery or even later In the more severe form the patient may find she cannot support herself on the affected leg Less severe forms may reveal themselves as pain or numbness or parasthesia on the affected side, and the patient may notice that she drags her toe She may, or may not, complain of backache The gait usually shows a decided limp, and there is *obvious drop foot on the affected side The muscle groups most commonly affected are the dorsiflexors of the foot and toes and also the peroneal muscles However, careful testing will often reveal weakness of other muscle groups,* such as the quadriceps, and this should alert the obstetrician to the possibility of a spinal disc lesion Some sensory loss will be found, usually over the dorsum of the foot and lower leg, sometimes corresponding to the cutaneous distribution of the peroneal nerve

TREATMENT

Initially the patient should be rested in bed and given suitable analgesia until the pain has been relieved The foot should be properly braced or splinted to prevent plantar flexion deformities This may be achieved by a plaster back slab or, more easily, by pillows and sandbags The pain is usually gone in 2–3 days and physiotherapy should then be initiated, first massage and passive *movements, then galvanic stimulation, and later active movements*

As soon as there is some active movement in the affected muscles, the patient should be allowed out of bed and encouraged to exercise It is helpful at this stage to get a built up shoe, which will make walking easier A toe spring, to help dorsiflex the foot, may also be attached

Some patients, in whom the disorder may have been caused by a prolapsed disc, require nursing in a plaster jacket and later postural exercises to strengthen the paravertebral muscles In a small proportion, neurosurgical intervention may be indicated

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Emergencies in the Newborn

Chapter 12

Timothy C. F. O'Connor, Denis Cavanagh

What different dooms our birthdays bring!
For instance one little mannikin thing
Survives to wear many a wrinkle
While death forbids another to wake
And a son that it took nine moons to make
Expires without even a twinkle
Thomas Hood (1798–1845)
Miss Kilmansegg Her Birth

The most common neonatal emergency is depression of respiratory, cardiovascular, and metabolic functions of the newborn However, other emergencies caused often by birth trauma, congenital malformation, or infection may arise in the immediate neonatal period and can be of particular concern to the obstetrician

NEONATAL DEPRESSION

Even in an adequately equipped unit with full time pediatric cover, the obstetrician will be occasionally called upon to resuscitate an unexpectedly depressed neonate It is very important, therefore, to have an organized plan of campaign to deal with such infants

ETIOLOGY

This may be caused by a large number of factors (Fig 12-1)

PREVENTION

The prevention of neonatal emergencies begins in the prenatal clinic and continues throughout pregnancy, labor, and the immediate postnatal period The following points are of particular importance

Pregnancy

Good prenatal care with particular attention to maternal nutrition and early detection of developing complications is essential

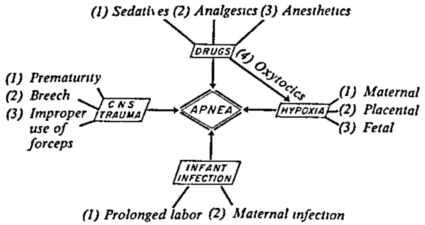


FIG 12 1 Causes of apnea or hypopnea in the newborn (American Academy of Pediatrics Resuscitation of the Newborn Infant 1958)

Labor

1. Term Careful observation for, and early treatment of, fetal distress, together with judicious use of analgesia and anesthesia are most important
2. Preterm The management of premature labor has been dealt with in Chapter 9 It suffices to repeat here that premature delivery should, if possible, be undertaken only where adequate facilities for management of the preterm infant are available

Delivery

The day of the traumatic high forceps delivery is gone but careful evaluation of the indication for and the type of forceps delivery is very important before proceeding to a midcavity forceps delivery

In addition, the following simple measures should be carried out at delivery

1. The mouth and nostrils should be gently suctioned on delivery of the infant's head *Vigorous* suctioning of the oropharynx is to be avoided since it may produce laryngospasm
2. The baby should be kept head down and the shoulders and trunk delivered slowly to enhance drainage of secretions
3. The infant should be toweled dry and rapidly removed to a heated environment

Immediate Postpartum Period

As pointed out by DeVore (1976) the best way to be prepared for a depressed neonate is to assume that all neonates will be depressed and be prepared to deal with all of them

MANAGEMENT

It is not always possible to prevent the delivery of a hypoxic infant, but the harmful sequelae can often be prevented by rapid efficient resuscitation

Equipment

A resuscitation table with attached radiant-heat source should be available so that procedures may be carried out without exposing the infant to cold Airways, face mask with pressure-release mechanism and inflation bag, infant laryngoscope, infant endotracheal tubes, oxygen, and suction apparatus with suitable catheters should all be readily available In addition, umbilical catheters, infusion solutions of sodium bicarbonate, sterile water and dextrose, should be available The drug naloxone (Narcan) should also be at hand As with all equipment, it is not only necessary that it be present, it is also necessary that it be in working order, regular checks of the equipment and solutions should be made

Assessment of the Neonate

The traditional method of evaluation of the neonate is the Apgar score (Table 12-1) It should be estimated 1 min after delivery of the infant (*not* 1 min after clamping and cutting the cord) and again at 5 min A neonate scoring 7–10 is considered normal, a score of 3–6 is moderately depressed, and a score below 3 indicates severe depression

TREATMENT OF MILD TO MODERATE DEPRESSION (APGAR 3–6) Once the airway has been cleared, place the mask over the infant s face, attach it to an oxygen supply, and inflate the bag The mask must be placed firmly over the face, but the chin should be elevated at the same time to allow free tracheal flow This is usually sufficient treatment and the infant will usually begin to breathe spontaneously, but if spontaneous respirations are not established and the heart rate falls below 100, intubation may become necessary

TREATMENT OF SEVERE DEPRESSION (APGAR 0–2) The same procedure should be followed, but if there is no response (change in color, increase in heart rate) in 30 sec, the infant should be intubated and the lungs inflated with oxygen (Fig 12-2 to 12-4) If the heart rate remains below 100, closed chest cardiac massage should be performed with two fingers over the sternum at a rate of 100/min If these measures do not result in a rapid response, the following should be done

1. Check if air entry is present and equal on both sides of the chest Absence of air entry means the tube is in the esophagus or pneumothorax is present bilaterally

TABLE 12-1 The Apgar Score*

Rating	0	1	2
Heart rate	Absent	<100	>100
Respiration	Absent	Hypoventilation gasping	Vigorous cry
Muscle tone	Limp	Some flexion	Spontaneous movement
Reactivity	None	Grimace	Cry
Color	Pale or blue	Body pink extremities blue	Pink all over

*To be performed exactly 1 and 5 min after complete birth of the infant

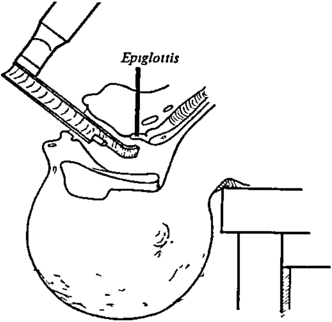
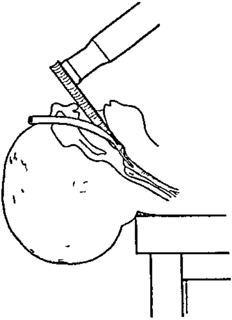


FIG 12-2 Oro-endotracheal intubation of the newborn The infant laryngoscope is used to elevate epiglottis (Modified from Hershenson Obstetrical Anesthesia Springfield, CC Thomas, 1955)

FIG 12-3 Vocal cords are visualized and infant endotracheal tube is passed between them



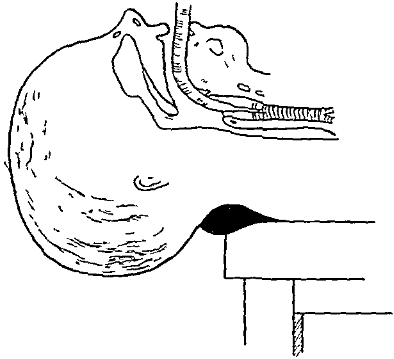


FIG 12-4 Laryngoscope is removed and endotracheal tube is left in place Secretions are aspirated and pure oxygen given

If there is unilateral absence of air entry, the tube should be withdrawn slightly (it may be in one or other main stem bronchus) If absent air entry persists, then there is either a pneumothorax or a diaphragmatic hernia on that side

2. If narcotics are suspected as the cause of depression naloxone (Narcan) 5 mg/kg body weight should be administered It may be given intramuscularly, and its effects will be seen in about 90 sec The effects of naloxone are of relatively short duration and may wear off before the effects of the narcotic do, after about 1 hour; the infant can become depressed again
3. The umbilical vein should be catheterized and sodium bicarbonate 2 mEq/kg body weight infused (~ 2 ml 7 5% $NaHCO_3$, or dilute 5 ml 8 4% $NaHCO_3$ in 5 ml 5% dextrose in water and infuse 10 ml in a term infant) The pH and blood gases should be estimated immediately and further infusions given if necessary

The essential points in infant resuscitation are

Clear the airway
Administer oxygen
Keep the infant warm
Be gentle

RESPIRATORY DISTRESS IN THE IMMEDIATE NEONATAL PERIOD

RESPIRATORY DISTRESS SYNDROME

This acute disorder of the premature infant which arises shortly after delivery is caused by a lack of surfactant in the lungs It should be suspected when a premature infant exhibits grunting respirations with indrawing of the intercostal spaces and sternum Chest x-ray is diagnostic The disease requires immediate skilled pediatric care in a neonatal intensive care unit

MECONIUM ASPIRATION

Respiratory distress and bilateral pneumothorax caused by inhalation of particulate meconium is a significant cause of death in term and post term infants (Claireaux, Fraser, Marshall, 1960) The incidence of this form of respiratory distress may be as high as 57% (Ting, Brady, 1975) unless the trachea is immediately suctioned at birth Tracheal aspiration will reveal meconium below the vocal cords in 56% of these infants, even after the onset of spontaneous respirations (Gregory, *et al*, 1974) Both of the latter two studies showed a marked decrease in the incidence of respiratory distress in infants in whom tracheal aspiration was carried out It is strongly recommended that all infants born through thick pea soup meconium have immediate endotracheal intubation and aspiration If meconium is found below the vocal cords, the infant should be admitted to the neonatal intensive care unit and observed for at least 12 hours

PNEUMOTHORAX

This problem may be suspected during resuscitation by unilateral absence of air entry The diagnosis is confirmed by chest x-ray films If the distress is increasing and there is radiologic evidence of mediastinal shift insertion of a needle and aspiration of air from the pleural cavity may provide temporary relief while skilled pediatric management is awaited

DIAPHRAGMATIC HERNIA

When severe respiratory distress is accompanied by a scaphoid abdomen with audible bowel sounds in the chest, diaphragmatic hernia should be suspected These infants require immediate surgery, and supportive measures are important while preparations are made

ESOPHAGEAL ATRESIA WITH TRACHEOESOPHAGEAL FISTULA

This disorder may cause respiratory distress in the early neonatal period Inability to pass a gastric catheter is diagnostic The treatment consists of surgical repair

CONGENITAL HEART DISEASE

Congenital heart disease may present as respiratory distress in the neonatal period The diagnosis may be suggested by cardiomegaly or the presence of a murmur, but definitive diagnosis should be made by a cardiologist

CHOANAL ATRESIA

Respiratory distress that is relieved by insertion of an oral airway should arouse suspicions of choanal atresia, a rare congenital malformation, in which a membrane blocks the passage of air through the nose to the nasopharynx Treatment is surgical, and an oral airway should be kept in position while awaiting operation

CONGENITAL PNEUMONIA

Respiratory distress in an infant whose amniotic sac has been ruptured for a prolonged period of time is suspicious of congenital pneumonia The mechanism of infection has already been discussed (see Ch 4, Life Threatening Infections) The diagnosis is often difficult, and there may be coexistent systemic infection Immediate transfer to a neonatal intensive care unit is indicated

OTHER CAUSES OF RESPIRATORY DISTRESS

Hemorrhage or perforation of a hollow viscus may also produce respiratory distress Rare congenital lesions of the larynx and trachea (atresia and stenosis) may also present this way In the case of laryngeal atresia, prompt tracheostomy may be life saving

BIRTH INJURIES

SOFT-TISSUE INJURIES

Cephalhematoma

Caused by a localized extravasation of blood between periosteum and skull, cephalhematoma presents as a cystic mass that does not cross suture lines (Fig 12 5) Skull x-ray films are mandatory because there is an underlying skull fracture in 25% of cases (Kendall, Woloshin) Surgical drainage is contraindicated unless infection supervenes Left alone, the cephalhematoma may be resorbed slowly or it may be converted into bone

Other Soft-Tissue Injuries

Edema of the scalp (caput succedaneum) or lips, nose, and eyelids in a face presentation may look alarming but will usually resolve without any treatment Soft tissue damage to the ears, nose, and mouth may occur during difficult deliveries but are rarely a cause for serious concern

Any eye injury, however, should be promptly seen by an ophthalmologist,

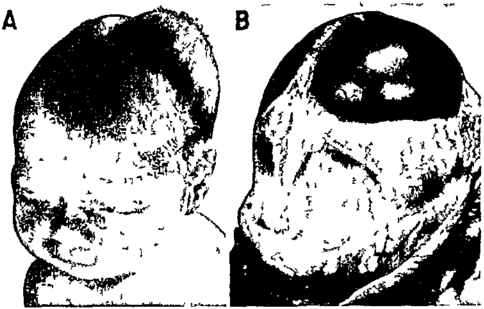


FIG 12 5 A Cephalhematoma over left parietal bone B Same patient with cephalhematoma exposed (Potter EL Pathology of the Fetus and the Infant, 3rd ed Chicago Year Book, 1961)

especially such serious ones as avulsion, corneal laceration, or retinal detachment

BONY INJURIES

Fractures of the Skull

Linear fractures of the parietal bone may occur, and in addition, skull depressions of the frontal or parietal bones may be caused by pressure of the fetal head against the maternal pelvis or may be associated with forceps delivery These are not necessarily true fractures (Greenhill 1974), and the skull often regains its normal contour spontaneously, but in true fractures operative elevation may become necessary

Fractures of the Vertebrae

Vertebral fractures are rare except where excessive force has been used Two situations in which fractures or dislocations of the vertebral column are likely to occur are shoulder dystocia and breech delivery The case for cesarean section in the breech with an extended head has been made in Chapter 9 The importance of such injuries lies in the accompanying damage to the spinal cord

Direct causes of spinal cord injury at birth are injurious forces at time of delivery (Towbin, 1964)

Traction on spinal axis of fetus causing stretch injury of vertebral column, cord and brain stem structures

Flexion of spinal column, excessive in degree, causing stretch injury and compression
Torsion of spinal column causing stretch injury
Contributing factors are as follows (Towbin)
Intrauterine fetal malposition, brow, face, breech, presentation
Dystocia
Prematurity
Primiparity
Precipitous delivery
Intraspinal vascular occlusive phenomena in fetus
Vertebral and foramen magnum malformations in fetus
The clinical effects of neonatal spinal cord and brain stem injury (Towbin, 1964) are as follows
Sudden death of fetus during labor (stillbirth) or directly after
Short survival of infant
- Respiratory depression at birth, shallow respiration, apnea
- Neurologic symptoms, "spinal shock", limp and pale appearance
- Pulmonary complications of hyaline membrane disease, pneumonia

Long term survival of infant
- Spinal cord injury, neurologic sequelae
  - Transient paralysis
  - Permanent paralysis (paraplegia tetraplegia)
  - Spasticity (clinically mild or latent)
- Spinal nerve root injuries, neurologic sequelae
- Brain stem injury
  - Cranial nerve deficits
- Cerebral hypoxic devastation (secondary)
  - Motor defects of cerebral palsy
  - Mentation deficits
  - Epilepsy

Fracture of the Clavicle

This injury is not uncommon, particularly during the delivery of big babies, breeches, or when shoulder dystocia is present Treatment is not necessary unless there is displacement, in which case the shoulders may be braced Recovery is usually good

Fracture of Long Bones

Fractures of the femur and humerus are becoming rare with the disappearance of both internal version and breech extraction Treatment is orthopedic

VISCERAL INJURIES

Traction on the trunk of a breech may cause rupture of the liver or spleen The trunk, of course, should never be handled during breech delivery In addition, overvigorous resuscitation may cause any of the injuries associated with such maneuvers in the adult

NERVE INJURIES

Facial Palsy

Facial palsy is usually associated with forceps delivery The nerve exits from the stylomastoid foramen and is protected in the adult by the styloid process, but in the neonate this process is not yet present, and the nerve is vulnerable to pressure, both here and as it crosses the ramus of the mandible The facies are very characteristic (Fig 12-6), and rapid recovery is the rule The eye on the affected side, however, should be protected from drying and ulceration of the cornea by regular application of a suitable ophthalmic solution

Brachial Palsy

The brachial plexus can be damaged by extreme lateral traction of the head in cases of shoulder dystocia or, in breech presentation, by traction downward on the shoulders in delivery of the aftercoming head The fifth and sixth cervical roots are most vulnerable, subsequent paralysis of the upper arm produces the clinical picture of Erb's palsy, in which the affected arm is rotated inward and held in a position of extension and adduction in the "porter's tip" position (Fig 12-7) When the seventh and eighth cervical and first thoracic roots are damaged the forearm and hand are affected If only the lower roots are damaged, only the hand is affected, and the lesion is known as Klumpke's paralysis Since the fifth cervical nerve root also supplies the diaphragm, respiratory difficulties may be encountered in some infants with a brachial plexus lesion A clinical picture similar to that of Erb's palsy may be produced by injuries to the shoulder joint

The diagnosis and treatment should be prompt The affected muscles should be rested and immobilized by flexing the arm above the infant's head and pinning it to the mattress cover so that the arm is abducted flexed, and externally rotated Physiotherapy is commenced after a few weeks, and a light splint may then be applied The typical brachial plexus palsy is associated with lacerations of the nerve sheath, edema, and hemorrhage, unless the nerve fibers are severed, the prognosis is excellent, with recovery occurring from 1 month to 2 years after birth

INTRACRANIAL HEMORRHAGE

Most of the fatalities during the perinatal period occur in association with cerebral birth injuries, and a considerable number of the severe injuries are attributable to intracranial hemorrhage Intracranial hemorrhage may result from injury, anoxia, or a combination of the two It is more likely to occur in a small baby or in a very large baby Tears of the tentorium cerebelli or falx cerebri are extremely rare in prematures whereas in mature infants they are a common result of cerebral birth trauma In prematures, the intracranial lesions seen most frequently are intraventricular and subarachnoid hemorrhages, they are the result, not of trauma, but of anoxia Regardless of the cause, the clinical picture is similar during the first day of life

Mortality from cerebral injuries has been reduced by about 50% in the past 30 years, mainly as a result of the reduction in the incidence of prolonged labor by the judicious use of oxytocin, forceps delivery, and cesarean section,

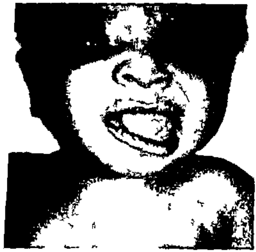


FIG 12-6 Right-sided facial palsy, demonstrating absence of right nasolacrimal fold, lack of facial muscle activity and failure of eye to close (Potter EL Pathology of the Fetus and the Infant, 3rd ed Chicago Year Book 1961)

FIG 12 7 Erb s palsy resulting from injury of fifth and sixth cervical roots of brachial plexus (Courtesy of Dr Ralph V Platou. Potter EL Pathology of the Fetus and the Infant, 3rd ed Chicago Year Book 1961)

and the reduction in the number of difficult midforceps deliveries, internal versions breech extractions and indeed all breech deliveries

During the descent through the birth canal, the head is compressed and the intracranial thickenings of the dura mater, called the falx cerebri and the tentorium cerebelli form a protective system of ligaments that resist excessive molding of the fetal head When molding is too rapid as in precipitate delivery, the tentorium or the falx may be overstretched and may rupture (Fig. 12 8) The more serious hemorrhage occurs from Galen's vein which, though only about 1 cm long is unsupported up to its point of entry into the straight sinus

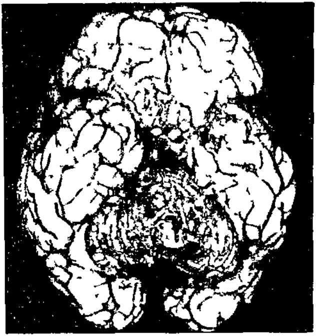


FIG 12-8 Ventral aspect of brain, showing hemorrhagic discoloration and ragged surface erosion of cerebellum in areas positionally related to margins of foramen magnum in a term infant delivered with high forceps that slipped and were reapplied. After birth, infant's respiration was delayed for 23 min, artificial respiration was maintained but infant died after 4 hours Autopsy revealed spinal injury as well as cerebellar damage (Towbin A Arch Pathol 77 620, 1964)

Rupture of this vein, or of the sinus itself, results in subdural or subtentorial hemorrhage with fatal compression of vital centers Although this injury may occur in association with an apparently easy, spontaneous delivery, it most commonly follows traumatic delivery of the aftercoming head in a breech presentation or a difficult midforceps delivery

In the presence of severe hemorrhage, the baby is either stillborn or in a state of asphyxia pallida The Apgar score of these babies is usually 4 or less both at 1 minute and at 5 minutes The baby who survives shows signs of cerebral irritation with restlessness, irritability, and a whimpering cry The anterior fontanella may bulge, and generalized rigidity is not uncommon When marked bulging of the anterior fontanelle is present, a subdural hematoma may be suspected, and aspiration is then indicated

CONGENITAL INFECTIONS

Figure 12-9 shows a number of agents that have been incriminated in congenital infection Detailed description of most of these is beyond the scope of this book but a brief discussion of a few is appropriate (see also Ch 4)

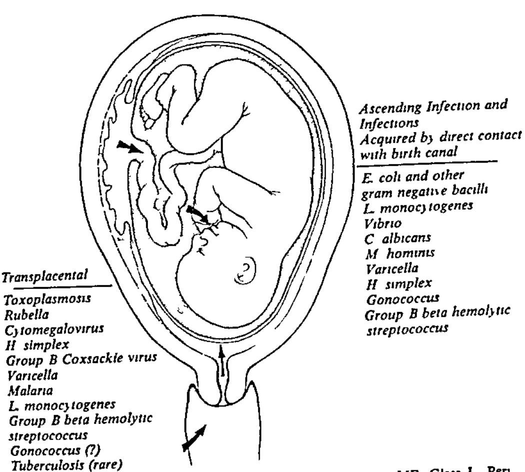


FIG. 12.9 Probable routes of fetomaternal infection. (Evans ME, Glass L: Perinatal Medicine. Hagerstown: Harper & Row, 1976)

SYPHILIS

The tragic results of untreated syphilis in pregnancy are well known. Although still very rare, this disease shows some evidence of resurgence (Oppenheimer, Hardy, 1971; Wilkinson, Heller, 1971). As well as the standard VDRL or rapid plasma reagin (RPR) test done in early pregnancy, patients who are suspected of being at high risk for syphilis should have a repeat test in late pregnancy (Center for Disease Control recommended treatment schedules 1976). Patients with a documented history of adequate treatment in the past need not be retreated unless there is evidence of reinfection (dark field positive lesion or fourfold rise in titer). Any patient treated in pregnancy should have monthly serologic tests during the remainder of the pregnancy.

GONOCOCCAL INFECTION

Although most obstetricians routinely screen for gonorrhea at first visit, a repeat screen should be obtained in the third trimester in patients at high risk of contracting the disease. The fact that the gonococcus may cause ophthalmia

neonatorum is well known, and eye prophylaxis with silver nitrate or antibiotic solutions has virtually eliminated this disease However, recent reports indicate that the gonococcus may also cause systemic infection in the neonate (Handsfield *et al*, 1972) and scalp infection (JAMA 1975) A further report suggests a higher incidence of unsuspected gonorrhea in patients with premature rupture of the membranes than in the general pregnant population (Handsfield *et al* 1973) These observations underline the wisdom of repeat screening for *Neisseria gonorrhoeae* in late pregnancy

GROUP B HEMOLYTIC STREPTOCOCCUS

A particularly virulent form of neonatal septicemia has been associated with the group B hemolytic streptoccus In its more acute form there is rapid onset of respiratory distress, pneumonia, and coma The mortality may be as high as 70% A less acute form with delayed onset is also described The incidence is thought to be about 1 500 live births (Franciosi *et al* 1973) and the infection is acquired by direct contact with the birth canal The incidence of vaginal colonization with this organism in the pregnant woman is 6% (Finch *et al* 1976), considerably higher than the neonatal infection rate, and this discrepancy excludes maternal treatment as an efficient method of prophylaxis This discrepancy may be explained, at least in part, by the findings of Baker and Kasper (1976), who noted that pregnant carriers of the bacteria, whose offspring developed the disease were deficient in antibody to some types of the group B streptococcus At the present time there does not appear to be an indication for routine prophylactic treatment of a patient found to be harboring this organism

HERPESVIRUS HOMINIS TYPE II

Although transplacental passage of this virus may cause fetal infection ascending infection during parturition is of greatest concern to the obstetrician (Nahmias *et al*, 1971) The virus may produce disseminated infection in the neonate, a condition with a mortality as high as 80%

Active genital herpes in late pregnancy is an indication for cesarean section, provided the membranes have not been ruptured for longer than 4 hours Unfortunately, 40–50% of maternal infections may be asymptomatic and therefore overlooked However, most of the asymptomatic lesions are cases of recurrent infection and it has been pointed out that neonatal infection is more common in patients with primary infection (Bolognese *et al*., 1976)

HEMORRHAGIC DISEASE OF THE NEWBORN

The fetus obtains its vitamin K via the placenta, but after delivery this source is not available and since the sterile neonatal gut requires time and bacteria to synthesize vitamin K, the blood clotting factors dependent on this vitamin fall after delivery, so that in approximately 1 300 infants a hemorrhagic diathesis results

This usually presents on the second or third day of life, when the infant shows signs of localized or generalized bleeding An enlarging cephalhematoma,

hematemesis, melena, or bleeding from the cord stump are all signs of this disorder The administration of 1 mg vitamin K_1 (Mephyton) will prevent the disorder It is important not to give water-soluble vitamin K, as large doses of this substance may cause hemolytic anemia and hyperbilirubinemia

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Chapter 13

Anesthetic Emergencies

Somporn N. Praphat, Denis Cavanagh

"Our very hopes belied our fears
Our fears our hopes belied
We thought her dying when she slept
And sleeping when she died"
Thomas Hood (1798–1845)
The Death Bed Stanza 3

Of every 100 maternal deaths in the United States, 6–10 are directly related to anesthesia Ninety percent of these deaths are preventable and result from unwise choice of anesthetic, improper technique, and excessive dosage Not infrequently the tragedy is twofold In addition to maternal and fetal mortality, the morbidity is considerable from prolonged fetal and maternal hypoxia The mortality and morbidity from this cause will only reach the irreducible minimum when obstetric departments are given 24-hour coverage by experienced and obstetrically oriented anesthesiologists Because of the popular misconception that obstetric anesthesia is minor anesthesia, the responsibility is largely delegated to nurse anesthetists and obstetric house staff This practice is at odds with the guidelines laid down by the Joint Commission on Accreditation of Hospitals, which states 'The same competence of anesthesia personnel shall be available for obstetric and emergency procedures as is available for elective procedures"

The obstetrician should realize that when an anesthesiologist is not available, the bulk of the responsibility for the prevention and management of anesthetic emergencies falls squarely upon the obstetrician's shoulders With a view to preventing anesthetic emergencies, the methods of obtaining pain relief in the obstetric patient is reviewed

For the relief of pain in labor three groups of agents are used

Analgesic agents alleviate pain
Anesthetic agents abolish pain
Amnesic agents reduce the patient's ability to remember pain

If an ideal anesthetic were available, it would relieve all pain, be nontoxic to the mother and fetus, cause no delay

labor, and yet allow the mother to remain cooperative throughout The desirability of alleviating pain during labor and delivery is obvious, because pain itself is debilitating to the mother However, all analgesics and anesthetics are potentially dangerous Irreparable damage can be done by them in a few seconds, and the following simple precautions should be taken before any form of anesthesia is given

1. Take a brief history of the present pregnancy and past illnesses Perform a brief physical examination with special attention to the patient's heart and lungs and to the presence of loose teeth and dental plates Never neglect to ask the patient when she has last eaten These simple steps will facilitate the choice of anesthetic
2. Obtain, beforehand, a thorough knowledge of the drugs to be used In particular, know the range of dosages, the advantages, limits, dangers, and allergic manifestations associated with each
3. Treat each case on its own merits and avoid the slavish enforcement of routine
4. Administer atropine sulfate, 0 4–0 6 mg, subcutaneously intramuscularly, or intravenously, unless it is specifically contraindicated, before the induction of general anesthesia
5. Give the minimum dosage to achieve adequate anesthesia, whether general or regional anesthesia is used
6. Keep at hand, in the delivery room pharmacologic antagonists to the drugs used
7. Make no promises to any patient that labor will be completely painless
8. Avoid oversedation of the patient, for this is dangerous to both mother and fetus There is no placental barrier to most drugs given to the mother for analgesia or anesthesia
9. Resist the overdemanding patient in all aspects of analgesia and anesthesia
10. Check the adequacy of the equipment available before an anesthetic is given

Suggestions Concerning Anesthesia

The following general suggestions are offered to the untrained anesthetist

1. Spinal anesthesia is contraindicated in the presence of recent hemorrhage, anemia, essential hypertension, and severe eclamptogenic toxemia with hypovolemia
2. Avoid the use of inhalation or intravenous anesthesia in the presence of a full stomach or a respiratory infection Sodium thiopental (Pentothal) is given in the minimum anesthetic dose in the presence of known or suspected heart disease and severe anemia
3. Use as small a concentration as possible of local anesthetic agents for infiltration Never use more than 1% procaine (Novocaine) and preferably use a 0 5% solution for cesarean section No more than 1 5 g procaine should be used, with epinephrine, 1 200 000 solution, given with it to prolong effectiveness in the area and reduce the absorption rate into the general circulation

THE CHOICE OF ANALGESIA

The amount of sedation required for each patient differs, so before routine sedation is prescribed, many factors must be considered Of particular interest are the health of the mother and the maturity of the fetus When the gestational age is less than 37 weeks, sedation must be kept to a minimum When the mother is healthy and the baby judged to be at term, a program of the following type is satisfactory in most primigravid patients

IN VERY EARLY LABOR *Before pain is a feature, 100 mg pentobarbital* (Nembutal) given intramuscularly will allow the patient to rest This may be repeated if necessary Particular care should be taken to avoid overdosage because no pharmacologic antagonist to the barbiturates is as yet available

WHEN PAIN BECOMES A DISTURBING FEATURE At this point the patient's cervix is usually about 2 cm dilated and well effaced Meperidine (Demerol), 50–100 mg, and promethazine (Phenergan), 25 mg, are given intramuscularly

If labor is prolonged it may be necessary to give another 50 mg of meperidine and 25 mg of promethazine

If the patient is delivered within 2 hours of receiving meperidine or morphine, naloxone (Narcan) should be given according to the following dosage schedule

1. To the mother Give 0 4 mg (1 ml) naloxone for each 100 mg meperidine or each 15 mg morphine received
2. To the infant If the baby appears to be narcotized, give 0 01–0 02 mg naloxone, pediatric preparation, into the umbilical vein at the time of delivery As pointed out by Ngai *et al* (1976), naloxone has a short duration of action

AMNESIA

Scopolamine (hyoscine hydrobromide), 0 4 mg subcutaneously, may be given in early labor, but it may cause troublesome hyperexcitability and should be avoided in overanxious patients In general the drug should be used with caution in patients with heart disease

THE CHOICE OF ANESTHESIA

Selecting the appropriate anesthesia will depend on three main factors 1) the condition of the mother, 2) the status and maturity of the baby, and 3) the experience of the anesthetist

Until anesthesiologists are available to give all types of anesthetics, the obstetrician will remain responsible for the anesthetic that the patient receives There is no such thing as a minor anesthetic, so all must be given with care If called upon to make a choice of anesthetic, the type with which the obstetrician is most familiar

Regional anesthesia is the method of choice for most deliveries "Balanced" anesthesia (sodium thiopental, muscle relaxant, and nitrous oxide) is usually

best for general anesthesia Although excellent results may be obtained in selected obstetric cases from the use of halothane, this should only be used under the supervision of a trained anesthesiologist, because of the tendency to uterine atony with postpartum hemorrhage

Every poor-risk patient deserves expert supervision, and priorities should be such that this is accomplished

LOCAL ANESTHESIA

Pudendal nerve block is the safest type of anesthesia for delivery and should be used in all cases, particularly when the baby is premature Low forceps delivery can usually be carried out quite satisfactorily, and in cases of assisted breech delivery, the nerve block can be supplemented with nitrous oxide and oxygen

Pudendal Nerve Block Anesthesia for Vaginal Delivery

Pudendal nerve block is best carried out between pains when the head is on the perineum with the patient in the lithotomy position, prepared and draped for delivery

1. With a fine hypodermic needle and a 0 5% procaine (Novocaine) solution, raise bilateral wheals in the skin midway between the anus and the ischial tuberosities In an obese patient the tuberosity may not be palpable so the gluteal fold is used as a guide
2. Insert the index and middle finger of the left hand into the vagina and the left ischial spine is palpated
3. While the fingers are kept in this position in the vagina, insert a 10-cm long 20 gauge spinal needle through the subcutaneous wheal on the left side and advanced to a point just posterior and cephalad to the ischial spines Inject 10 ml of a 0 5% procaine hydrochloride solution, but before the injection is made, it is essential to aspirate to make certain that the tip of the needle is not in a blood vessel This occasionally occurs, in which case the needle is simply withdrawn somewhat and reinserted, however, following delivery the area of the ischial spine should be examined occasionally (rectal examination is satisfactory) to make certain that, as occurs in rare instances, a hematoma does not develop
4. Withdraw the spinal needle to a point just below the skin surface and directed laterally and anteriorly toward the ischial tuberosity Inject anesthetic solution (5 ml) at this point.
5. Withdraw the needle to a point just below the skin and direct it anteriorly to deposit about 5 ml of solution in the left labial region just below the skin
6. Withdraw the needle and direct it posteriorly, depositing about 5 ml of solution posterior to the anus to block the nerve fibers of the fourth sacral nerve
7. Infiltrate the perineal body with about 5 ml of solution
8. Repeat the procedure (step 2 through 7) on the right side, with the right index and middle fingers placed in the vagina to identify the ischial spine
9. It is occasionally necessary to infiltrate bilaterally about 1 inch above the clitoris, to block fibers of the ilioinguinal nerve and dorsal nerve of the clitoris (Fig 13-1)

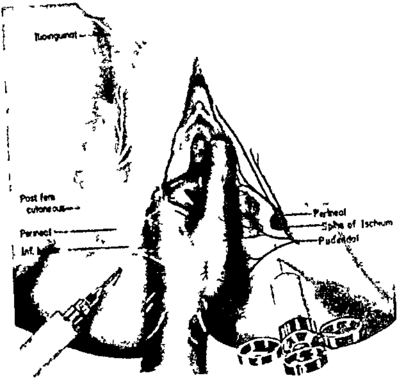


FIG 13 1 Pudendal nerve block (transperineal) Procedure is used to block nerve supply to pelvic floor (Courtesy of L A Bunim Am J Obstet Gynecol 45 805, 1943 Copyright CV Mosby Co)

Transvaginal Pudendal Nerve Block

When sufficient room is available between the baby s head and the vaginal wall, the needle is passed through the sacrospinous ligament and about 10 ml of 0 5% procaine solution is injected in the region of the ischial spine (This is done bilaterally) Before any injection in this region, aspiration must be carried out Good perineal anesthesia will usually be obtained by a single injection in the region of each ischial spine as described here, but some prefer to supplement this by labial and perineal infiltration The use of a needle guide such as the Iowa trumpet ensures more accurate placement of the local anesthetic, and reduces the possibility of damage to the head of the fetus or the rectum of the mother

Paracervical and Uterosacral Block

The terms paracervical and uterosacral block should be used interchangeably, because the effect is identical Mepivacaine (Carbocaine), 1%, gives a duration of action of 60–90 min Lidocaine (Xylocaine), 1%, is also satisfactory, but its duration of action is approximately 45 min Epinephrine, 1 200,000, may be added to the lidocaine to increase its duration of action This may produce

uterine inertia, but with an oxytocin infusion, normal progress in labor can be maintained

With regard to paracervical block, a warning note was sounded by Nyirjesy *et al*, who used the technique on 68 patients and reported that in 15 there was evidence of fetal depression attributable to the anesthesia itself They also noted that the frequency of bradycardia was reported to be from 3 7 to 20% on review of the literature, and that reported maternal complications included convulsions in three patients, sacral neuritis in two patients, suspected broad ligament hematoma in one patient, and peripheral vascular collapse in another With the single injection method of paracervical block, babies have died as a result of the inadvertent injection of a bolus of local anesthetic into the fontanelles

Another limitation of the method is that delivery must occur within the duration of action of the drug when the single-dose technique is used A method of continuous paracervical block has been described by Baggish This was begun in some primigravidas at less than 2 cm dilatation in the presence of an established pain pattern He suggested the use of specially made 20 gauge, 9 5-in Teflon needles with Teflon and stainless steel stylets

The technique is as follows The patient is taken to the delivery room and is placed in the lithotomy position Skin wheals are raised bilaterally at a point midway between the ischial tuberosity and the anus With the stainless steel stylet in place, the needle is inserted through the skin of the perineum and directed along the lateral wall of the vagina (Fig 13-2) The needle enters the vagina in its upper third and then repierces the mucosa of the lateral fornix penetrating the parametrium to a depth of about 1 cm The Teflon needle is fixed to the perineum with a 3–0 chromic catgut suture, and the steel stylet is removed The needle is aspirated to ensure that a blood vessel has not been entered, and about 10 ml of 1% mepivacaine is injected The Teflon stylet is then placed in the needle, and the patient returned to her bed The needle is left *in situ*, and injections are made as necessary every 40–60 min

Burchell and Sadove have also described a method of continuous paracervical block

This form of anesthesia is relatively easy to use, produces a minimum of patient discomfort, allows maximum patient cooperation, and reduces the need for respiratory depressant drugs This continuous technique appears to be particularly applicable to the management of premature labor The routine use of paracervical block remains controversial, but certainly it is a useful technique in selected cases

Local Infiltration Technique for Cesarean Section

Local anesthesia has been largely replaced by conduction anesthesia, but when a skilled anesthesiologist is not available, it still has a place The surgeon stands at the right side of the patient and proceeds in the following manner

1. Prepare and drape the abdomen as usual and make a skin incision (Fig 13-3)
2. Use a 20-gauge, 0 75-in long needle to raise a wheal in the region of the umbilicus (Fig 13-4)
3. Charge a large syringe with freshly mixed 0 5% procaine solution, to be injected through a 4-in 20 gauge needle Make the intradermal injections in

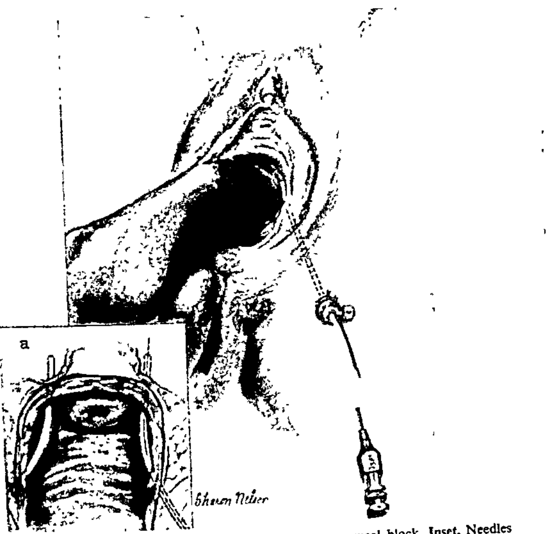


FIG. 13-2. Method of administering continuous paracervical block. Inset. Needles pierce the upper, lateral vagina, inserting 1 cm adjacent to the external os and 1 cm deep into the parametria. (From Baggish MS: Am J Obstet Gynecol 88:968, 1964)

several stages all the way down to the symphysis pubis. The subcutaneous injections extend outward about 3 cm on each side of the midline. Unless the area is highly vascular, the solution is injected continuously as the needle is advanced (Fig. 13-5).

4. Make the incision through the skin and subcutaneous tissue after approximately a 3-min wait (Fig. 13-6).
5. Inject local anesthetic into the exposed anterior rectus sheath in a manner similar to the method used for skin infiltration. More anesthetic is used in the suprapubic area than elsewhere (Fig. 13-7).
6. Incise the anterior rectus sheath after a wait of about 3 min for the anesthetic to take effect. Separate the rectus muscles to expose the posterior fascia and peritoneum (Fig. 13-8).

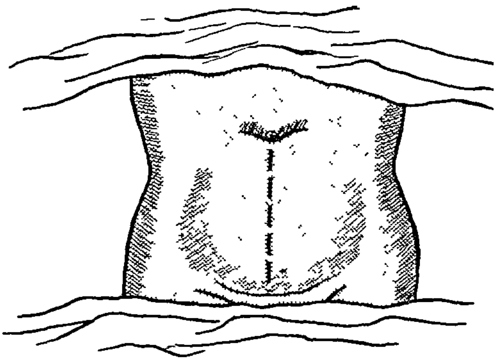


FIG 13-3 Local infiltration anesthesia for lower uterine segment cesarean section Site of incision (Redrawn from Greenhill JP Obstetrics, 12th ed. Philadelphia, WB Saunders, 1960)

FIG 13-4 An intradermal wheal is raised by using a fine short needle, just below the umbilicus (Redrawn from Greenhill JP Obstetrics, 12th ed Philadelphia, WB Saunders, 1960)

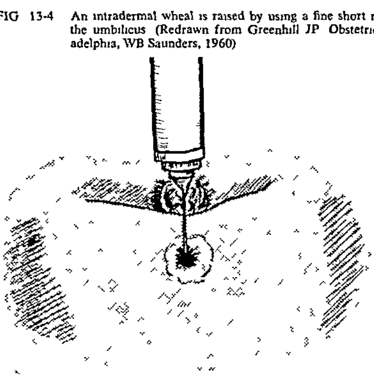


FIG 13 5 A larger and longer needle is used to inject the 0 5% procaine *solution subcutaneously* (Redrawn from Greenhill JP Obstetrics, 12th ed Philadelphia, WB Saunders, 1960)

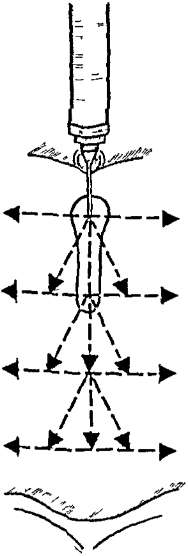
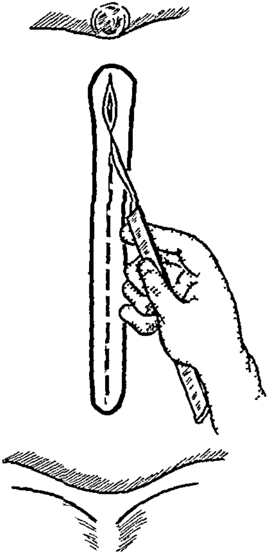


FIG 13-6 After a wait of about 3 minutes *skin and subcutaneous fat* are incised (Redrawn from Greenhill JP Obstetrics, 12th ed *Philadelphia WB Saunders, 1960)*



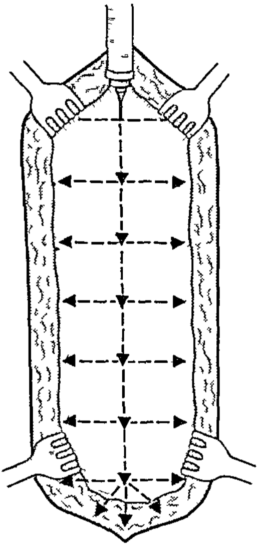


FIG 13 7 Anterior rectus sheath is infiltrated with special attention to suprapubic area (Redrawn from Greenhill JP Obstetrics 12th ed Philadelphia WB Saunders 1960)

7 Carefully infiltrate the posterior fascia and the peritoneum with local anesthetic in a manner similar to that used for the fascia (Fig 13 9)
8 Pick up the posterior fascia and peritoneum with fine hemostats incised and enter the peritoneal cavity (Fig 13 10)
9 Retract the wound gently but sufficiently to give adequate exposure Avoid intraabdominal packs if possible Inject about 10 ml of the anesthetic solution into and under the loose peritoneum of the bladder reflection (Fig 13 11) This also aids in the later dissection of the bladder flap and the bladder
10 Massage the raised peritoneum to spread the solution downward and laterally (Fig 13 12)

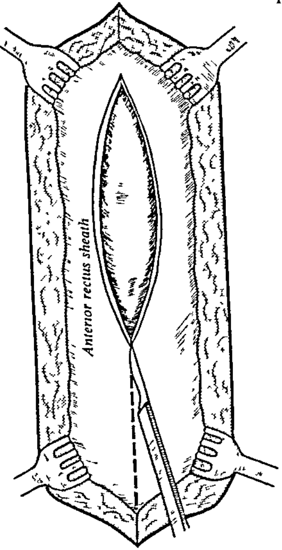


FIG. 13-8. After a 3-minute wait anterior rectus sheath is incised, and rectus muscles are separated to expose posterior fascia and peritoneum. (Redrawn from Greenhill JP: Obstetrics, 12th ed Philadelphia, WB Saunders, 1960)

11. Incise the loose bladder flap of peritoneum transversely with Metzenbaum scissors. Use the index finger to push down the bladder.
12. Make a small transverse incision in the lower uterine segment and enlarge it by finger traction or by using bandage scissors. Take care not to traumatize the uterine vessels.
13. Deliver the baby through the incision in a manner similar to that in vaginal delivery. (If oriented in this manner, the operator will rarely have difficulty in delivering a baby at cesarean section using the hand or one blade of the standard obstetric forceps.)
14. Allow the placenta to separate spontaneously if possible, give 0.2 mg of methergine intravenously as soon as the baby is delivered.
15. Remove placental fragments and pieces of membrane with a gauze sponge covering the fingers, and close the uterus in layers in the usual manner.

The peritoneum, fascia, subcutaneous tissue, and skin can usually be closed without further infiltration.

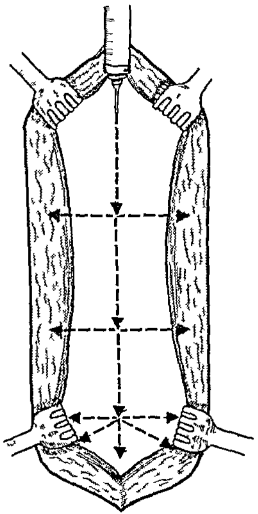


FIG 13 9 Posterior fascia and peritoneum are carefully infiltrated (Redrawn from Greenhill JP Obstetrics 12th ed Philadelphia, WB Saunders 1960)

If cesarean hysterectomy is to be carried out, further infiltration will be required and general anesthesia is preferable

MAJOR CONDUCTION ANESTHESIA

The parturition pain pathways are diagramed in Figure 13 13 The techniques of spinal caudal and lumbar epidural anesthesia are well described in the literature The principles of conduction analgesia and anesthesia are diagramed in Figure 13 14 Drugs such as procaine and lidocaine, injected into the subarachnoid or extradural spaces exert a regional anesthetic effect and then pass into the mother s bloodstream where they are rapidly disposed of Although the breakdown products pass the placental barrier readily they are apparently relatively innocuous to the fetus When cesarean section is to be performed and the baby is premature conduction anesthesia should be used unless a specific contraindication is present

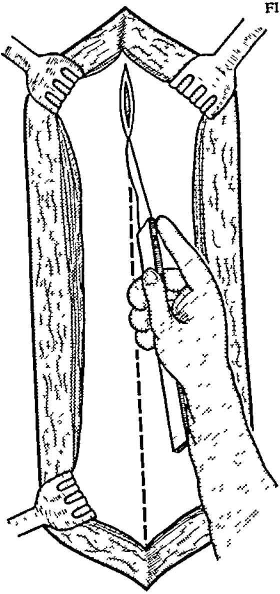


FIG. 13-10. Posterior fascia and peritoneum are incised, and peritoneal cavity entered. (Redrawn from Greenhill JP: Obstetrics, 12th ed. Philadelphia, WB Saunders, 1960)

The main dangers in the use of these drugs are hypotension, respiratory depression, and convulsions These manifestations are the result of patients' receiving too much drug too fast.

Pregnant women are more likely to have cardiovascular collapse during conduction anesthesia than are patients undergoing surgery. The pregnant uterus compresses the vena cava and reduces venous return At the same time the venous pressure rises in the communicating veins of the vertebral column and is transmitted to the subarachnoid space, resulting in increased spread of the anesthetic agent. Flattening of the normal lumbar lordotic curve further heightens the level of anesthesia. There is also increased sympathetic tone in the lower extremities during pregnancy, and this is lost under conduction anesthesia.

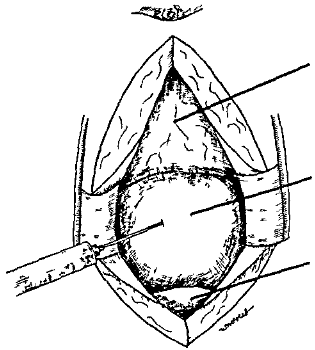


FIG 13 11 Procaine solution (0 5%) is injected into and under the loose peritoneum over lower uterine segment (Redrawn from Greenhill JP Obstetrics 12th ed Philadelphia WB Saunders 1960)

Although an experienced anesthesiologist may elect to use conduction anesthesia on a patient with eclamptogenic toxemia the undertaking is hazardous in the hands of an amateur because of attendant hypovolemia Hypotension is especially liable to occur following large doses of magnesium sulfate Oxygen must be immediately available and an intravenous infusion of 1 liter of balanced salt solution must be begun prior to conduction anesthesia A centrally acting vasopressor drug such as ephedrine 50 mg given intramuscularly 10–15 min prior to administration of conduction anesthesia is effective in preventing the onset of hypotension The anesthetic dosage should be less than that for the average patient of the same height and weight who is undergoing surgery

During subarachnoid block the anesthetic solution should be injected slowly between contractions while the patient is in the left lateral position She should then be placed immediately in the supine position with the head raised on a pillow, and the table tipped to the left side The advantage of the left lateral displacement of the uterus is evident (Fig 13 15) When the patient has to be supine a method of sustained left uterine displacement should be used (Fig 13 16) Good relaxation of the abdominal wall and anesthesia to T-7 can usually be obtained using 6–8 mg of tetracaine (Pontocaine) in 2 0 ml of hyperbaric solution

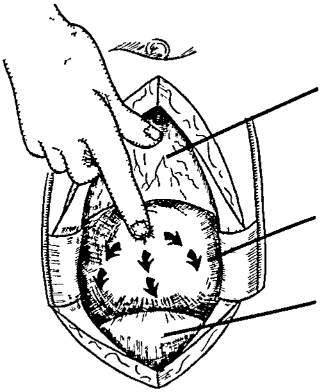


FIG 13 12 Raised peritoneal reflection is massaged as shown, to spread anesthetic solution downward and laterally (Redrawn from Greenhill JP Obstetrics, 12th ed Philadelphia WB Saunders 1960)

Low Spinal Anesthetic

This type of anesthesia has a "saddle' distribution and is widely used for low forceps and breech delivery For this type of distribution, the patient remains seated for 2 min after the injection The injection is given to a count of 1-and-2 and-3) After being seated for 2 minutes, the patient is placed in the recumbent position with a pillow under her head Her blood pressure must be taken every 30 sec for at least 5 min following the administration of any type of spinal anesthesia

For hyperbaric spinal anesthesia, a safe maximum single dosage for vaginal delivery is procaine hydrochloride, 30–75 mg or lidocaine, 25–50 mg, or tetracaine, 2–5 mg diluted with 10% dextrose in water, to make 1 5 ml of solution for injection

CAUDAL AND EPIDURAL ANESTHESIA Anesthesia of these types require a trained team and are generally not available in smaller hospitals The patient must be carefully observed throughout administration, because inadvertent subarachnoid injection may result in total spinal block, with respiratory arrest and severe hypotension leading to cardiac arrest The technique for standard epidural block is presented in Figure 13-17 Local anesthetics for spinal anesthesia in vaginal delivery, with dosage and duration are presented in Table 13-1

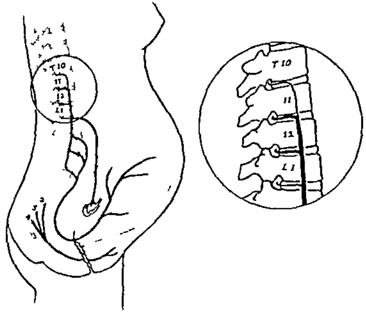


FIG 13 13 Parturition pain pathways The uterus including cervix, is supplied by sensory (pain) fibers that pass from uterus to spinal cord by accompanying sympathetic nerves in the following sequence uterine cervical and pelvic plexuses hypogastric nerve superior hypogastric plexus lumbar and lower thoracic sympathetic chain and thence through white rami communicantes and posterior roots The primary pathways (inset, thick lines) enter 11th and 12th spinal segments while secondary auxiliary pathways enter at T10 and L1 Pathways from perineum pass to sacral spinal cord via pudendal nerves (Modified from Bonica JJ Principles and Practices of Obstetric Analgesia and Anesthesia Philadelphia Davis 1967)

GENERAL ANESTHESIA

General anesthesia is usually unnecessary for normal vaginal delivery, but it may be indicated for midforceps or breech delivery and internal version as well as for cesarean section It should not be used for vaginal delivery when the fetus is normal and under 37 weeks Whether given intravenously or by inhalation, anesthetic agents reach the fetus in about 2 min Generally the depth of fetal depression is proportional to the depth of maternal anesthesia

Probably the greatest hazards in general anesthesia are aspiration of vomitus with respiratory obstruction, aspiration pneumonitis, and cardiac arrest The stomach empties very slowly during labor, and when the mother has eaten within 12 hours, general anesthesia should be avoided If it is absolutely necessary, it should be administered by a skilled anesthesiologist, with endotracheal intubation of the patient while she is awake Even in the absence of food, the stomach contains gastric secretions, and with a pH less than 2 5, as little as 50 ml can cause fatal aspiration pneumonitis (Mendelson's syndrome) When internal version is necessary and the uterus is poorly relaxed, halothane (Fluothane) anesthesia is most effective for producing adequate

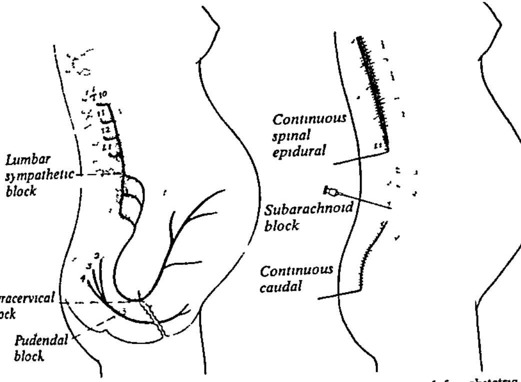


FIG 13 14 The most common regional anesthetic techniques used for obstetric analgesia anesthesia (Modified from Bonica JJ An atlas on mechanisms and pathways of pain in labor What s New 217 16, 1960)

uterine relaxation When deep anesthesia of any type is given, artificial ventilation of the baby should be provided after delivery The choice of anesthesia depends upon 1) the experience of the anesthetist available, 2) the condition of the mother, 3) the general condition and maturity of the fetus, and 4) the time that has elapsed since the ingestion of food by the mother

Ideally, endotracheal intubation should always be carried out, and the following procedures* should be instituted

Preparatory procedures
1. Antacid, *e g*, 30 ml magnesium trisilicate ½ hr before induction
2. Preoxygenation
3. Atrophine, 0 4 mg intravenously
4. *d* Tubocurare, 3 mg intravenously

Induction
1. Sodium thiopental, 2.5 mg/kg body weight, intravenously
2. Cricoid pressure
3. Succinylcholine, 80 mg intravenously
4. Avoidance of positive pressure
5. Endotracheal intubation
6. 0 2% succinylcholine intravenous infusion

* Adapted from Bonica JJ Clinics in Obstetrics and Gynaecology Philadelphia, WB Saunders, 1975

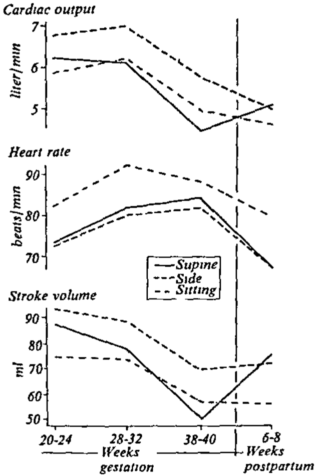


FIG 13-15 Effect of posture on maternal cardiac output, heart rate, and stroke volume during different periods of gestation (Ueland K et al Am J Obstet Gynecol 104 856, 1969)

The sequence of general (balanced) anesthesia for emergency vaginal delivery is given. The preparatory phase is performed just before the patient goes to surgery and is continued during the preparing and draping of the patient Induction of anesthesia should be started only when all the obstetric team is ready to deliver Constant communication between obstetrician and anesthetist is required

Agents for General Anesthesia in Most Common Use

NITROUS OXIDE Nitrous oxide and oxygen anesthesia is unsatisfactory for the obstetric patient. The high concentration of nitrous oxide (85%) required to produce total anesthesia causes quite marked maternal and fetal hypoxia This type of anesthesia is particularly undesirable in black women in whom a hemoglobinopathy has not been excluded, because hypoxemia may precipitate a sickle-cell crisis

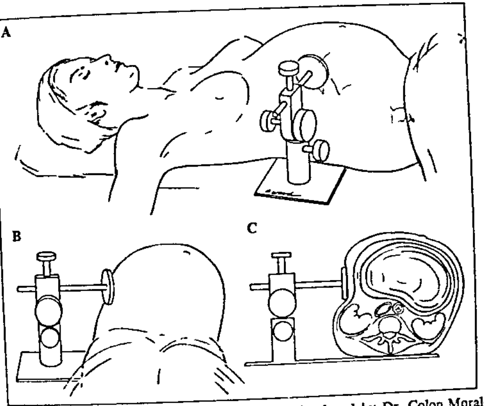


FIG 13 16 Left uterine displacement device developed by Dr Colon Morales of Puerto Rico A. Lateral view of parturient on operating table with the device fixed to the table, and its arm with displacer fixed against parturient s abdomen B Inferior anterior view C. Schematic cross section shows how device relieves compression on inferior vena cava and aorta by displacing uterus and its contents to the left

A concentration of 60% nitrous oxide with 40% oxygen gives adequate and safe analgesia during late first-stage and second-stage contractions The mixture should be given at the onset of and continued throughout each contraction but should be discontinued between contractions This method is useful when spinal saddle block anesthesia proves to be inadequate or when supplementation of pudendal nerve block or local infiltration anesthesia is required

The various machines designed for the self administration of "gas and air" analgesia are rather unsatisfactory because the oxygen concentration in the mixture is too variable for safety

SODIUM THIOPENTAL This ultra short-acting barbiturate is administered intravenously Its primary use in obstetrics is in the induction phase of balanced general anesthesia for cesarean section Preoxygenation of the patient for 3–5 min before induction is well worth while, and if she has not been premedicated, she is given 0 4–0 6 mg atropine intravenously Also, curare, 3 mg, is given intravenously in order to reduce intragastric pressure and minimize fasciculations The patient is meanwhile prepared and draped for the operation Sodium thiopental is then given intravenously in a dosage of 4 mg/kg body

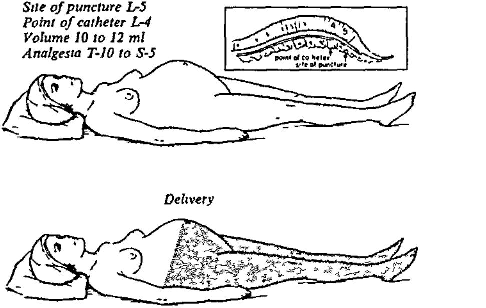


FIG 13 17 Technique for standard epidural block for labor and vaginal delivery A continuous catheter is inserted through a needle placed at fifth lumbar interspace and advanced so that its point is at level of fourth lumbar vertebra **Top.** Light stippling shows analgesia extending from T_{10} to S_5 **(Box)** achieved with low concentrations of local anesthetics **Bottom** Just prior to delivery a higher concentration is injected to produce perineal relaxation and anesthesia (Bonica JJ [ed] Obstetric Analgesia and Anesthesia Heidelberg, Springer-Verlag, 1972)

weight, and 80–100 mg succinylcholine (Anectine) is given intravenously to facilitate endotracheal intubation A mixture of nitrous oxide (60%) and oxygen (40%) provides an adequate level of anesthesia The dosage of sodium thiopental should not exceed 250 mg intravenously prior to the delivery of the baby, and in the presence of hypotension or hypovolemia the dosage should be reduced by half Muscle relaxation is maintained with a continuous infusion of 0 2% succinylcholine It is now generally accepted that the longer the induction–delivery interval, the higher the incidence of low 1-min Apgar scores So the shorter the induction–delivery interval, in concert with good surgical technique, the less will be the depression of the newborn

HALOTHANE One of the new group of halogenated inhalation anesthetics, halothane is especially useful because it produces rapid uterine relaxation, facilitating such procedures as version and extraction and manual removal of the placenta However, its use requires a skilled anesthetist, because deep planes of anesthesia must be achieved rapidly Initially, 2% halothane with 40% nitrous oxide and 60% oxygen is used, and if uterine relaxation is achieved before loss of consciousness occurs, the halothane is reduced to 0 5%, and then discontinued as soon as the procedure is completed A cuffed endotracheal tube is inserted if 2% halothane must be continued beyond a few

TABLE 13-1 Dosage and Duration of Local Anesthetics for Spinal Anesthesia in Vaginal Delivery

Drug	Dosage (mg)	Total volume (ml)	Duration* (min)
Procaine	75	1 5–2	45– 60
Tetracaine	5	1 5–2	90–120
Lidocaine	37 5	1 5–2	75– 90
Mepivaca ne	37 5	1.5–2	80–110
D buca ne	3 75	1 5–2	120–180
Propitocaine	37 5	1 5–2	80–110

*Duration can be increased 40–50% by adding 0 3 mg epinephrine or 3 mg phenylephrine to the solution

(Bonica JJ Obstetric analgesia anaesthesia Recent Advances and current status Clinics in Obstetrics and Gynaecology Philadelphia WB Saunders, 1975)

minutes Halothane is a potent vasodilator and myocardial depressant, so hypotension may occur Postpartum hemorrhage is common following the use of halothane, and methergine is more effective than oxytocin in controlling bleeding after halothane administration

MUSCLE RELAXANTS Drugs such as succinylcholine may be combined with general anesthesia if further voluntary muscle relaxation is required

In pregnancy there is a decrease in serum pseudocholinesterase, and so succinylcholine is not hydrolyzed as efficiently as in the nonpregnant woman A 'block-aid' monitor should therefore be used, to avoid dual block from excessive succinylcholine administration

ANESTHETIC EMERGENCIES

ESSENTIAL EQUIPMENT FOR DEALING WITH ANESTHETIC EMERGENCIES

The following equipment should be available and functioning in every delivery room

1. A supply of oxygen
2. Means of supplying intermittent oxygen at pressures of 20–40 cm H_2O for infant resuscitation
3. A well fitting face mask with attached rebreathing bag and preferably a clear plastic face piece
4. A set of pharyngeal (size III, IV, and V) and nasopharyngeal airways
5. Two laryngoscopes (adult and newborn size) and a set of endotracheal tubes (size 32, 34, and 36) with a malleable metal stylet available for each
6. A suction apparatus provided with adequate connections and suction tips, as well as a large stomach tube

7. A sterile set of tracheostomy instruments and tubes (size 5 or 6)
8. A cardiac defibrillator near at hand immediately accessible
9. Drugs Several ampules of the following drugs should be available
   A. 10% calcium chloride solution
   B. Epinephrine (Adrenalin), 1 10,000 and 1 1000 solutions
   C. 1% lidocaine solution 50–100 mg intravenously as a bolus
   D Amobarbital or thiopental for intravenous use
   If an unduly large amount of procaine, etc , is used for regional anesthesia, a soluble barbiturate should be mixed and kept ready in a syringe for im mediate use in case convulsions occur
   E. Sodium bicarbonate, 44 mEq (50 ml)
   F. Ephedrine sulfate (25 mg/ml) or mephentermine (Wyamine) (30 mg/ml)
   G. Isoproterenol (Isuprel), 1 mg in 500 ml 5% dextrose in water
   H. Naloxone, adult, 0 4 mg/ml, pediatric 0 02 mg/ml
   I. Succinylcholine, 20 mg/ml
   J. Lidocaine, given in infusion in a dosage of 1–4 mg/min
   K. Procainamide (Pronestyl), given in an infusion in a dosage of 1–3 mg/min for maintenance purposes
   All regional anesthetic drugs should be in clearly marked ampules These should be autoclaved and never stored in an antiseptic solution, as the ampule may crack and contamination may occur The name of the drug and dosage on each ampule must be checked and rechecked before it is given to the patient
10. A supply of sterile syringes and needles of all sizes
11. Plasma, 25% serum albumin, or dextran These must be available immediately, and whole blood and fibrinogen within a few minutes
12. Tubes (heparinized, oxalated, and nonoxalated) available for collecting blood samples
13. Delivery room tables and beds These should be narrow, equipped with restraints and should be suitable for immediate conversion to the head down position Preferably the table should be equipped with a block to give sustained left uterine displacement

PRECAUTIONS

All patients should have an intravenous infusion started before any anesthetic is given Balanced salt solution is recommended, approximately 500 ml should be given through a plastic cannula prior to induction of anesthesia If these simple precautions are taken, the obstetrician will have provided the patient with a lifeline and so will be in a much better position to deal immediately with any emergency situations that may arise

ACUTE RESPIRATORY PROBLEMS

Pharyngeal Relaxation with Obstruction by the Tongue

This condition should be recognized in a patient whose respiratory move ments become increased and irregular The marked diminution of ventilation is reflected in the movements of the breathing bag and in progressive cyanosis All types of respiratory obstruction must be considered seriously, for hypoxia is an important factor in the development of cardiac arrest

A respiratory obstruction of this type is most commonly due to the tongue falling back and becoming approximated to the posterior pharyngeal wall The cause of obstruction can usually be recognized from the loud stertorous respiration

The condition can be prevented by inserting a pharyngeal or nasopharyngeal airway

MANAGEMENT

1. Turn the patient's head to one side, and apply pressure at the angle of the mandible to push the jaw forward With this action the stertorous sounds usually cease and the patient's breathing returns to normal
2. Insert a pharyngeal airway of the proper length (If masseteric spasm prevents the insertion of a pharyngeal airway, gently insert a nasal airway)
3. Give oxygen until the patient's color is good and breathing is regular The anesthesia is then continued

Laryngospasm

Stimulation during light anesthesia such as that caused by the introduction of an oropharyngeal airway while the pharyngeal reflexes are still present, may induce laryngospasm Prevention consists essentially of avoiding the causes

MANAGEMENT This will depend largely upon the cause In general, the following routine should be followed

1. Eliminate the cause
2. Give oxygen under intermittent positive pressure in an effort to break the spasm
3. Give 40–60 mg of succinylcholine intravenously, unless the spasm is broken within 1 min This acts almost immediately, and after the administration of the muscle relaxant, positive pressure ventilation should be carried out until the muscle relaxant wears off and respiratory exchange is adequate The effect of this dose usually wears off 3–6 min after intravenous administration
4. If anesthesia is to be continued, then intubation should be performed

Everyone giving anesthesia should be able to pass an endotracheal tube This is much easier in some patients than in others and the patient must not be allowed to become hypoxic while the attempt is prolonged It is good practice for the beginner to hold his breath while attempting endotracheal intubation for this will make him mindful of the patient's hypoxic state If intubation is not achieved within 3 minutes, stop, oxygenate the patient, and try again

Aspiration of Vomitus

The aspiration of particulate matter is the most common cause of maternal death during general anesthesia, and a full stomach is the reason As many as 12–48 hours may be necessary for the stomach to empty during labor Thus all obstetric patients should be considered to have a full stomach

Ideally, an endotracheal tube should be inserted in all obstetric patients when general anesthesia is given

Aspiration of vomitus can also occur with high spinal anesthesia The patient may be unable to cough up vomitus refluxing into the pharynx from the

stomach Spinal anesthesia, therefore, does not necessarily protect the patient from the dangers of aspiration

The particulate matter will cause atelectasis and, perhaps ultimately, lung abscess formation The aspiration of gastric secretions also causes severe chemical pneumonitis (Mendelson's syndrome)

PREVENTION

1. Give atropine, 0 4–0 6 mg intramuscularly 45 min before the induction of anesthesia This will reduce intragastric pressure and so reduce the possibility of aspiration
2. Give curare, 3 mg 2–3 min before succinylcholine, to diminish the intragastric pressure rise and the fasciculations that follow administration of succinylcholine
3. Forbid all solid foods and limit oral fluids during labor
4. Do not use general anesthesia without endotracheal intubation Regional anesthesia should be used for patients who have eaten within 12 hours, unless it is contraindicated
5. If general anesthesia is essential for the delivery of a particular patient who has eaten within 12 hours, and no one is available to intubate the patient, insert a large caliber stomach tube The patient will then empty her stomach spontaneously as the tube is passed, or the contents can be aspirated once it is in place In addition, a cuffed endotracheal tube should be used during anesthesia The inflated balloon prevents aspiration, in the event that reflux of the stomach contents occurs despite precautions
6. Combat hypotension following spinal or epidural anesthesia This can result in loss of consciousness with accompanying loss of protective upper respiratory tract reflexes
7. Give magnesium trisilicate (or other antacid), 30 ml orally, about 30 min before induction to raise the pH of stomach contents If the pH of the stomach contents is lower than 2 5 and the patient aspirates, chemical pneumonitis will develop
8. Use pressure on the cricoid cartilage (Sellick maneuver) so that it is pushed cephalad and posteriorly to occlude the esophagus This pressure should be applied from the time the patient is asleep until intubation has been completed and the cuff inflated
9. Do not remove the endotracheal tube until the patient has regained consciousness
10. Before inducing general anesthesia, make certain than an ample supply of oxygen and a suction apparatus are available
11. Insert an arterial line to allow monitoring of blood gases

MANAGEMENT

1. Remove the anesthetic mask and turn the patient's head to one side At the same time have the table converted to a steep Trendelenburg position
2. Use a jaw retractor to force the jaw open, and insert the fingers of the right hand behind the patient's last molar tooth
3. Scoop out solid material with gauze around the fingers
4. Suction the pharynx rapidly with a large-caliber suction tip
5. Give oxygen under pressure to try to relax the associated laryngospasm If a satisfactory response is obtained by this time, the patient is turned to the semi-prone position and postural coughing encouraged

6. If respiratory distress develops, pass an endotracheal tube immediately and aspirate the tracheal contents. Ventilate the lungs with pure oxygen and repeat endotracheal suction until satisfied that the airway is clear. The addition of positive end expired pressure (PEEP) may aid oxygenation while allowing lower inspired oxygen concentrations, thus decreasing the danger of pulmonary oxygen toxicity.
7. If the pulse weakens or slows and cyanosis persists, perform a tracheostomy. Aspirate the trachea quickly and supply oxygen through a soft rubber catheter. If difficulty persists, solid material is still present in the bronchi, and bronchoscopy should be carried out without delay. If cardiac arrest occurs, perform closed chest cardiac massage.

POSTANESTHESIA CARE

1. Obtain an x-ray film of the chest immediately after breathing is restored.
2. Encourage postural coughing.
3. Observe the patient for clinical or radiographic evidence of collapse or pneumonitis, and treat as necessary.
4. If the pH of the material aspirated from the trachea is less than 2.5, or if wheezing and bronchospasm persist, assume that acid aspiration has occurred. This is a serious problem, even though there is no solid material in the patient's stomach. If acid aspiration has occurred, hydrocortisone, 200 mg, is given intravenously immediately, followed by 100 mg intravenously every 8 hours. This dosage is continued until the chest x-ray reverts to normal, for at least 24 hours, even if the chest film shows no significant changes. If bronchospasm occurs, give the patient a continuous intravenous infusion of aminophylline, 500 mg in 500 ml of 5% dextrose in water or an intravenous infusion of isoproterenol in balanced salt solution, starting at a rate of 1 μg/kg/min, increasing as necessary.
5. Give positive-pressure ventilation with sufficient oxygen to maintain Pa_{O_2} at 80–100 torr, the Pa_{CO_2} at 30–35 torr, and the pH at 7.4–7.5. If pulmonary edema develops, it should be treated with morphine, 10 mg, intravenously, and furosemide (Lasix), 40 mg, intravenously.
6. Give antibiotic coverage to these patients on the basis of a Gram stained smear and culture of the aspirate taken from the trachea. Ampicillin, 1 g every 4 hours intravenously is a good choice unless a contraindication is present.
7. Obtain an x-ray film of the chest every 12–24 hours. The right lung is more commonly affected than the left. Acid aspiration is indicated by soft, irregular mottling, and solid aspiration shows uniform densities with some degree of mediastinal shift.
8. Keep the patient in an intensive care unit for continuous monitoring and treatment. Give oxygen, a mechanical respirator should be used as necessary. Make blood gas determinations frequently, and see that a tracheostomy set is kept by the patient's bedside.

Respiratory Depression

By far the most common cause of respiratory depression is oversedation, but it may occur in association with poliomyelitis, myasthenia gravis, or multiple sclerosis.

PREVENTION Sedation must be kept to the minimum in all patients while reasonable comfort is maintained Oversedation reduces labor contractions and is not in the best interest of the patient or her baby

MANAGEMENT

1. Give naloxone (0 4 mg) intravenously for each 15 mg of morphine or 100 mg of meperidine received
2. Give oxygen and assisted respiration to ensure adequate pulmonary ventilation
3. Insert an endotracheal tube if necessary
4. Perform a tracheostomy if the respiratory rate is not being maintained above 8/min, if oxygenation is inadequate, if aspiration of secretion is required, and if the endotracheal tube is not satisfactory

CARDIOVASCULAR EMERGENCIES

HYPOTENSION

Severe hypotension from spinal anesthesia is the second most common cause of maternal anesthetic death This can occur with high spinal or improperly administered caudal or epidural anesthesia The basic problem is that the capacity of the vascular bed is suddenly increased so that relative hypovolemia is produced

Prevention

1. Hydration with balanced salt solution is important, give 500 ml over a 15-min period prior to spinal anesthesia
2. Administer all spinal anesthetics slowly, no matter what agent is used Dosage for spinal anesthesia in the pregnant woman should be approximately half that for the same patient in the nonpregnant state
3. Check the blood pressure every 30 sec for at least 5 min after a spinal anesthetic has been given
4. Avoid spinal anesthesia in women with essential hypertension or anemia and in women who have had recent bleeding

Management

Following spinal anesthesia the patient may become suddenly stuporous, cold and hypotensive Occasionally convulsions may occur The following steps should be taken

1. Increase the rate of intravenous infusion
2. Give ephedrine, 12 5 mg intravenously Repeat the dose in 1–2 min if no blood pressure response is observed
3. Elevate the patient's legs 90° This simple procedure makes about 700 ml of blood immediately available to the rest of the circulation The table should *not* be in the Trendelenburg position and two pillows must be kept under the patient s head and shoulders
4. If hypotension persists, turn the patient to the left lateral position or push

the uterus to the left, so that hypotension is not aggravated by compression of the inferior vena cava by the gravid uterus

5. Give oxygen by mask
6. If cardiovascular collapse occurs, it should be treated as described in chapter 3
7. If respiratory paralysis occurs as a result of high spinal anesthesia, give assisted or artificial ventilation

HYPERTENSION

Much less common than hypotension, hypertension may be caused by 1) oxytocic drugs given to toxemic patients who are hypovolemic, or 2) overdosage with vasopressor drugs given prophylactically with spinal anesthesia

Prevention

Avoidance of the causes and the careful observation of all patients under anesthesia can prevent hypertension Frequent blood pressure readings are essential in patients under any type of anesthesia but are particularly important in patients under spinal anesthesia

Management

The causative agent should be discontinued and hypotensive drugs given if available Especially useful is hydralazine (Apresoline), 20–40 mg added to the intravenous fluid and titrated against the blood pressure

CARDIAC ARREST

Sudden failure of the pumping action of the heart will lead to an immediate reduction in available blood volume This can be caused by myocardial infarction, cardiac tamponade, or massive pulmonary embolism, but when it occurs in an otherwise healthy woman it is usually referred to as cardiac arrest A common cause of the latter is anesthesia

The general occurrence rate is now about 1 in every 1500 uses of anesthesia—between 5000 and 10 000 times annually in the United States The total number of obstetric patients affected is unknown, but because most of these are healthy young women, it occurs less frequently than among surgical patients When it does occur in obstetric patients, however, more than half the mothers and even more of the babies are lost

A period of arrest of more than *4 min* at normal body temperature is rarely compatible with complete recovery Prolonged cerebral hypoxia results either in death or survival in a decerebrate state

Predisposing Factors

1. Hypoxia at any time during anesthesia Hypoxia frequently exists in the induction stage of anesthesia, and this is the most common time for cardiac arrest in the obstetric patient.

2. Hypercapnia occurring during the course of an operation—sometimes evidenced by premature ventricular contractions or bradycardia Patients who receive large amounts of cold stored blood may develop severe bradycardia, and elevated serum potassium may contribute to the occurrence of arrest The blood must be warmed before administration because this drives potassium into the cells
3. Anesthetic agents in excessive dosage All such agents may cause cardiac arrest, especially if vasoconstrictive agents such as adrenalin or vasopressin are used to effect hemostasis in the operative field
4. Heart disease Cardiac arrest occurs above five times more commonly in women with heart disease
5. Rapid blood loss
6. Vagal stimulation, especially in the presence of hypoxia or hypercapnia, which may occur during such procedures as gynecography or laparoscopy

Prevention

1. Atropine, 0 6 mg subcutaneously, should be given beforehand to all patients having general anesthesia.
2. When hypercapnia is present, the patient should be given artificial ventilation to restore the blood to a pH over 7 4, Pa_{O_2} 80–100 torr, and Pa_{CO_2} less than 40 torr If hyperkalemia is present 20 g of glucose with 10 units of regular insulin will help to reduce the serum potassium level by driving the potassium into the cells
3. The stomach should be empty before general anesthesia is undertaken, for aspiration of vomitus may lead to cardiac arrest as well as asphyxia

Diagnosis

The most important factor in improving the survival rate of patients in association with cardiac arrest is early diagnosis Vascular instability—as evidenced by hypotension and abnormal cardiac rate or rhythm, respiratory difficulty, and muscular twitching—commonly precedes arrest The alert anesthesiologist or anesthetist will recognize these danger signs and prompt action will avert catastrophe The anesthesiologist must observe the vital signs in all patients carefully throughout anesthesia, and in obstetric cases he must not leave the mother unattended while resuscitating the baby The anesthesiologist must be especially alert during the induction stage of anesthesia and report immediately the absence of a carotid pulse or recordable blood pressure If these do not reappear within 1 min, then the occurrence of cardiac arrest should be considered established The diagnosis may also be made on the basis of the absence of heart sounds on auscultation and the absence of bleeding from the operative site The pupils are fixed and unreactive to light, but initially are not dilated

If at cesarean section neither aortic pulsation nor heart impulses can be palpated, external cardiac massage should be begun

The establishment and maintenance of adequate respiration are the responsibility of the anesthesiologist The establishment and maintenance of adequate circulation are the responsibility of the obstetrician For success in treatment,

teamwork is essential and all must be familiar with the steps to be taken There is no time for discussion

A cardiac resuscitation set should be available in every operating room and delivery room

Management of Established Cardiac Arrest

1. Stop the administration of any drug that may have precipitated arrest
2. *Clear the airway of mucus or other material Give pure oxygen and artificially* ventilate the lungs at a rate of 20 times per minute If the patient is not already intubated, a pharyngeal airway or an endotracheal tube is inserted as soon as the color of the mucosa improves If no mask or oxygen supply is available, mouth to mouth breathing is instituted in an effort to inflate the lungs The chest should rise symmetrically with inflation, and this is confirmed by *auscultation If the stomach becomes distended, insert a nasogastric tube* and decompress it.
3. Lower the head of the table *slightly*, and raise the legs to increase venous return
4. Begin external cardiac massage with the patient in a shallow Trendelenburg position (Fig 13-18)

   Place the heel of the right hand, with the left hand on top of it, on the sternum just above the xiphoid Exert vertical pressure, using body weight, to push the sternum inward for a distance of about 1.5 in This action forces blood out of the heart into the lungs and the general circulation When the pressure is released, the heart fills Chest compression should be performed about 60 times per minute If no help is available, the patient is pressure-*ventilated twice for every 15 sec of external cardiac compression.*

An electrocardiographic tracing must be obtained If the tracing shows ventricular fibrillation rather than arrest in asystole, defibrillation should be carried out

Every obstetrician must be thoroughly familiar with the management of cardiac arrest, and a cardiac resuscitation set and *an electrical defibrillator* should be available *for every operating room* and delivery room In summary, the plan of action can be remembered by the use of the mnemonic **"STOP MD** and **Support."**

Stop drug—*e g* thiopental sodium

Trendelburg the patient and elevate the legs to 90° for 10 sec or longer if an *assistant is available*

Oxygen is given by mask (or endotracheal tube if in place)

Pressure ventilation is carried out at the rate of about 20 per minute If necessary, an endotracheal tube should be passed and assisted ventilation continued

Massage the heart at 60–80 times per minute by external compression if asystole is present

Defibrillate, with electric defibrillator *(DC 200–400 watt seconds) if electro*cardiogram shows ventricular fibrillation

Supportive measures should be undertaken—*e g* , compression of the aorta, and administration of fluids and inotropic drugs Sodium bicarbonate should be given for treatment of metabolic acidosis, 44 6 mEq (50 ml) immediately

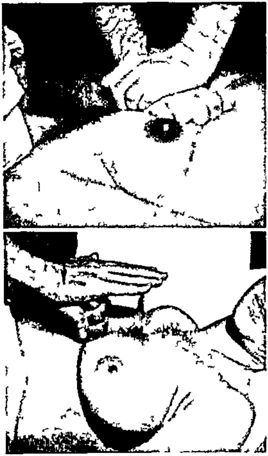


FIG 13 18 Technique of external cardiac massage (Top) Cardiac arrest Pat ent is placed on flat firm surface Vertical pressure is exerted using body weight to depress sternum 1 5–2 in toward vertebral column This action forces blood out of heart and into pulmonary peripheral circula tion When pressure is released cardiac filling occurs **(Bottom)** Cardiac arrest Patient is placed on flat firm surface Ulnar aspect of left closed fist with right hand on top of it is placed on lower third of sternum This affords more localized compression probably increasing efficiency and minimizing injuries Body weight is used to apply com pression as with Kouwenhoven s method (Cavanagh D DeCenzo JA Ferguson JH Obstet Gynecol 22 56 1963)

and at 10-min intervals thereafter if determinations of arterial pH and base indicate the emergency persists

If the heart persists in asystole give 0 5–1 ml epinephrine 1 1000 by the intracardiac route Also give calcium chloride 5–10 ml intravenously Ade quate fluids must be given and an infusion of metaraminol (100 mg in 500 ml) is useful for maintenance If there is a recurrence of ventricular fibrillation following conversion to normal rhythm procainamide, 10% solution 100 mg

intravenously should be given A maintenance infusion of 1% lidocaine solution, at the rate of 1–4 mg/min should be given as necessary.

Results

If adequate oxygenation and circulation are established within 4 min, approximately 50% of patients will recover

TOXICITY TO LOCAL ANESTHETICS

The most common cause of a reaction to local anesthetics is overdosage True allergic or hypersensitivity types of reactions are rare, especially with the newer amide local anesthetics such as lidocaine and mepivacaine The amount of local anesthetic used is not so important as is the blood level reached, and this will be high if inadvertent intravenous injection occurs Signs of local anesthetic toxicity are seen in pregnant women when blood levels of 5 μg/ml lidocaine or mepivocaine are reached Toxicity in the newborn exists with half this blood level

Factors that affect the rate of local anesthetic absorption are as follows

1. The total amount of local anesthetic used
2. The time over which the local anesthetic is administered
3. The method of administration Subcutaneous injection has the slowest uptake, intramuscular injection is more rapid, and topical application is more rapid still Intravascular injection results in the highest and most rapidly achieved blood levels This is most likely to occur during epidural block because the epidural veins are dilated during pregnancy
4. Vascularity of the area into which the local anesthetic is injected
5. The concentration of local anesthetic solution used
6. The use of vasoconstrictors (epinephrine) and spreading agents (hyaluronidase) that influence blood levels

Clinical Picture

Either stimulation or depression may occur

STIMULATION This may be *characterized by nausea and vomiting, hyperactivity with tinnitus, tremors, blurring of vision, and a feeling of impending doom Grand mal convulsions with associated apnea and laryngospasm may occur Hypertension and tachycardia may occur*

DEPRESSION A period of depression will generally follow a stage of stimulation Patients who have been heavily sedated with barbiturates may not manifest stimulation Lidocaine may initially produce somnolence and sedation before excitement If the blood level of the drug rises rapidly, as when intravascular injection occurs, the stimulation phase may not be seen

Management

1. Control hyperexcitability and convulsions Give only small doses of depressants because depression will follow The rapid, short-acting barbiturates such as sodium pentobarbital (Nembutal), 25–50 mg, should be given intra-

venously Diazepam (Valium) is satisfactory for postpartum use If convulsions are not controlled promptly, endotracheal intubation should be carried out with the aid of succinylcholine, 100 mg intravenously The patient is then ventilated with 100% oxygen

2. Treat cardiovascular depression by improving cardiac output and vascular tone with ephedrine, 12 5–25 mg intravenously, repeated as necessary If hypotension occurs, displace the uterus to the left, elevate the legs, and oxygen is given In addition, give a rapid infusion of 500 ml of a balanced salt solution If cardiac arrest occurs, treat it as described in the section Cardiovascular Emergencies in this chapter
3. Treat respiratory depression by ventilating the patient with 100% oxygen If the patient continues to convulse, short acting muscle relaxants, such as 50 mg succinylcholine, may be used to allow intubation If means of positive pressure ventilation and someone skilled in intubation are not available, muscle relaxants must *not* be used In addition, a barbiturate should be given to depress the cerebral convulsive focus
4. Combat gastric aspiration Upper airway reflexes are lost If no one is available to intubate, the patient should be placed on her left side in the head down position This will cause gastric contents to run away from the larynx and into the mouth, from which they can be suctioned
5. Combat effects on the fetus If the mother experiences a toxic reaction, the fetus must be monitored electronically, and scalp sampling may be useful Paracervical block is the most common reason for high fetal blood levels of anesthetic, but rarely is cesarean section indicated

THE SEVEN PILLARS OF PREVENTION

The blame for most deaths in obstetric anesthesia lies not in our stars but in ourselves " This is apparent on consideration of the most common causes of death, and the toll will be immediately reduced if seven simple suggestions are followed

1. *Do not* give general anesthesia unless you are sure that the patient s stomach is empty and you are competent in endotracheal intubation
2. *Do not* overdose with spinal anesthesia—*i e*, give smaller doses than those used in nonpregnant patients, and give them slowly
3. *Do not* use drugs that you are unfamiliar with as routine precautions may be omitted with resultant disaster—*e g* the administration of continuous caudal analgesia without adequate observation of the patient
4. *Do not* use incompatible drug combinations—*e g* the use of methergine after phenylephrine (Neo-Synephrine) administration resulting in acute hypertension or perhaps cerebral hemorrhage
5. When local anesthesia is required
   A. *Use* the minimal effective concentration of solution and
   B. *Always aspirate* before infiltration when there is any danger of inadvertent intravenous injection—*e g* during pudendal nerve block of paracervical block
6. *Ensure* that adequate oxygenation is maintained throughout anesthesia
7. *Deal promptly* with all emergency situations

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Medical Emergencies in Obstetrics

Denis Cavanagh

Chapter 14

"Death lies on her, like an untimely frost"
William Shakespeare (1564–1616)
Romeo and Juliet—Act IV, Scene 5

Medical emergencies of a nonobstetric nature can constitute threats to the life of the mother and her unborn child A number of these conditions such as clotting disorders (Chapter 2) cardiac arrest and pulmonary embolism (Chapter 3), acute renal failure (Chapter 5) and acute pyelonephritis (Chapters 3 and 4) have already been dealt with However, a number of medical emergenices require further consideration All these conditions are dealt with in detail in a number of medical textbooks, therefore, only those aspects relevant to their occurrence in the pregnant woman will be discussed and an outline of management given

HEART DISEASE

Heart disease is a major cause of maternal death in the United States, but if maternal disability is minimal both maternal and perinatal mortality are only slightly increased With marked degrees of heart disease, the maternal death rate is in the region of 2% and perinatal mortality may reach 50%

The functional capacity of the heart is the best single indicator of the patient's prognosis and the New York Heart Association Classification (1964) is a useful guide

Class I Normal physical activity causes no discomfort

Class II Normal activity causes discomfort and slight dyspnea

Class III Less than normal activity causes discomfort or dyspnea

Class IV Any physical activity is impossible, and the patient is dyspneic at rest

About 85% of maternal deaths in women with heart disease occur with class III or class IV disease, and patients in these two classes constitute only about 15% of those with heart

disease It is especially important to watch the patients who are pregnant for evidence of heart failure, and a mild respiratory infection may precipitate cardiac decompensation Because of the altered circulatory hemodynamics of pregnancy, left heart failure is more common than right heart failure in the pregnant patient, and the most common manifestation is acute pulmonary edema

ACUTE PULMONARY EDEMA

Although we are concerned primarily here with pulmonary edema resulting from preexisting heart disease (rheumatic or congenital), it may also occur in a patient with toxemia of pregnancy or even in a previously normal patient, as a result of fluid overload In the case of preexisting heart disease, the failure is most likely to occur at the time of maximum blood volume (28th to 36th week of pregnancy), during the second stage of labor, or in the third stage of labor when a sudden increase in blood volume occurs as a result of the decrease in the vascular space following delivery of the placenta and retraction of the uterus

Prevention

Appropriate medical and obstetric measures in the patients at risk will help to prevent pulmonary edema Appropriate obstetric measures are as follows

1. Give adequate analgesia Avoid scopolamine, and use morphine, 10 mg, in preference to meperidine
2. Use pudendal nerve block or conduction anesthesia for delivery
3. Give oxygen freely during labor and immediately postpartum
4. Shorten the second stage of labor by forceps delivery to save the patient the effort of bearing down
5. Unusual care should be taken to avoid excessive postpartum bleeding Unless there is significant postpartum hemorrhage oxytocics should *not* be given
6. If possible, deliver the patient in the left lateral position Avoid cesarean section unless a definite obstetric indication is present. If the patient is delivered in the lithotomy position, lower the legs promptly after delivery to reduce drainage of blood into the general circulation At that time, even a volume of 700 ml might be critical
7. Discourage the patient with class III or IV cardiac disease from breast-feeding her infant.
8. Keep the patient in the hospital for at least 1 week after delivery Give her appropriate advice with regard to contraception if she desires it Make appropriate arrangements for home help before the patient is discharged from the hospital

Diagnosis

This is readily made because of the onset of severe dyspnea The patient frequently has a coughing episode, and frothy pink fluid appears at her lips She is cyanotic and anxious Tachycardia is present, and the neck veins are usually distended

Treatment

The following procedures should be instituted

1. Place the patient in the sitting position with legs down
2. Give oxygen 100% by mask with mechanical positive pressure ventilation
3. Inject morphine 10–15 mg intravenously
4. Inject lanatoside C (Cedilanid), 0 8 mg intravenously, follow it with 0 4 mg every 6 hours for two doses
5. Slowly inject aminophylline, 0 5 g intravenously
6. Inject furosemide (Lasix), 40 mg intravenously
7. Carry out physiologic venesection, using tourniquets on three of four limbs and rotating them every 15 min
8. In an acute emergency, perform a phlebotomy and remove up to 500 ml of blood to reduce the load on the heart

When the acute episode is over, follow up therapy will include completion of digitalization with a maintenance dose of digoxin 0 25 mg daily, and placement on a low sodium diet An oral diuretic should be given if it is required For purposes of further care, any patient who develops heart failure should be considered henceforth to have heart disease class IV, and appropriate measures should be taken

ASTHMA

The pregnant woman who is having an acute asthmatic attack presents a very difficult problem The patient who is admitted in labor with bronchospasm should receive vigorous therapy for relief of symptoms Patients being treated for asthma should have this therapy continued throughout pregnancy and labor

Diagnosis

A history of previous asthmatic attacks related to allergy, bronchitis, or anxiety may be obtained

Physical examination reveals respiratory distress with inspiratory retraction, prolonged expiration, and prominent rhonchi diffusely over both lung fields

Laboratory findings in these patients may contribute little However, *in severe asthmatic attacks, arterial blood gases may show decreased arterial oxygen tension, an increase in arterial carbon dioxide tension, and acidosis*

Management

Even in the emergency situations treatment can be initiated during preparation for delivery

1. Start positive-pressure treatments, using epinephrine nebulization
2. Give a subcutaneous injection of 0 2 ml epinephrine (1 1000 solution) This is contraindicated for use with trichloroethylene, cyclopropane, or halothane anesthesia Alternatively, aminophylline, 250–500 mg in 500 ml 5% dextrose in water, may be given as an intravenous infusion
3. Give hydrocortisone, 100–200 mg, intravenously as an initial dose Follow this with 100 mg in a liter of 5% dextrose in water every 8 hours

4. If metabolic acidosis develops, correct it with 1 ampule (44.6 mEq) of sodium bicarbonate intravenously. Take care in administering sodium bicarbonate in the presence of a high CO_2 tension in arterial blood.
5. Give antibiotics if there appears to be an initiating or perpetuating infectious factor.
6. Administer oxygen liberally to the patient during labor to maintain good arterial oxygen tension.
7. Anesthesia for delivery should preferably be pudendal block, although a major conduction anesthetic given by an experienced anesthesiologist is very satisfactory. General anesthesia is best avoided in the obstetric patient with asthma.

SICKLE-CELL DISEASE

Sickle cell disease is a dominant hereditary disorder seen most commonly in black women. The term covers a number of hemoglobinopathies. Patients with the sickle cell trait (SA) are at little risk. Patients with sickle-cell anemia (SS) or patients with sickle-cell (SC) disease are in an entirely different category and pregnancy can be very dangerous for both mother and baby. In either of these two conditions a sickle cell crisis may occur.

SICKLE-CELL CRISIS

The problem is very serious. The maternal mortality ranges in various series from 0 to 25%, and the perinatal mortality ranges from 25% to 50%.

A crisis is manifested by attacks of headache, and abdominal, joint, or bone pains lasting from several hours to several days. The hemoglobin level remains relatively unchanged despite a hemolytic crisis. Numerous nucleated red cells may be present in the peripheral blood, and leukocytosis is almost invariable.

X-rays of the bones may reveal cortical thinning and diffuse osteoporosis with thickening of trabecular markings on x ray films.

The sickle-cell smear is diagnostic for both sickle-cell trait (SA) and sickle cell anemia (SS). Hemoglobin electrophoresis will identify other hemoglobinopathies such as SC disease and S-thalassemia.

Bone and joint pains resemble those of rheumatic fever. The presence of a rigid, tender abdomen may suggest an acute surgical emergency, but bowel sounds are normal. Headache, convulsions and paralysis due to cerebral thrombosis may be mistaken for a variety of conditions, including preeclampsia or eclampsia. Splenomegaly is a feature of SC disease but rarerly occurs in sickle cell anemia. In sickle-cell anemia the picture is often compounded by the presence of iron deficiency anemia.

Complications such as hematuria, pyelonephritis, osteomyelitis, leg ulcers and gallstones are not uncommon.

Management of Sickle-Cell Crisis

1. Give oxygen (100%) by face mask.
2. Give analgesia. Because of the severity of the pain, morphine sulfate 10 mg every 4 hours, may be required.

3. Correct acidosis, with sodium bicarbonate 1 ampule (44 6 mEq) added to intravenous fluids
4. Treat complications such as pyelonephritis
5. Consider giving a partial exchange transfusion

Indications for an exchange transfusion are a) painful crisis, b) pregnancy at 28 weeks with a previous clinical course indicating severe hemoglobinopathy, or c) *previous partial exchange transfusion with hematocrit under 25% or hemoglobin A less than 40%* The procedure involves a series of phlebotomies with each liter of blood removed being replaced with fresh, washed, packed cells from a hemoglobin AA donor, and the balance of the patient's blood volume being reconstituted with lactated Ringer's solution The phlebotomy and infusions may be performed alternately or simultaneously The plan of treatment as suggested by Schwartz is as follows

1. For every 500 ml of blood removed, infuse two units of packed red cells and 100–200 ml lactated Ringer's solution, with careful monitoring for hypotension, circulatory overload, and transfusion reaction
2. *Obtain a hematocrit after the third exchange transfusion is completed*
3. Repeat the procedure until an equilibrated hematocrit of 35% is reached Most patients will require phlebotomy of 1500 ml to achieve an adequate hemoglobin AA concentration and maximum benefit from the procedure
4. Follow-up should include a) serial hematocrits (should be maintained at greater than 25%), b) serial reticulocyte counts (this should remain at 0–1% to demonstrate marrow suppression), c) serial hemoglobin electrophoresis, to demonstrate hemoglobin A of greater than 40%, d) folic acid, 1 mg *per os* daily, e) iron therapy if indicated by serum iron studies, f) microscopic examination of urine for evidence of infection on each visit, g) *close monitoring of the patient for early signs of preeclampsia, to which these patients are predisposed, h) close monitoring for signs of chronic fetal distress, in view of the* high perinatal mortality associated with this condition

DIABETES MELLITUS

Diabetes mellitus occurs as a complication of one in every 300 pregnancies in the United States Although maternal deaths are now rare because of improved treatment, the fetal mortality remains in the region of 20% Pregnant diabetics are classified as follows

Class A Diabetic status based only on an abnormal glucose tolerance test

Class B Onset of diabetes after age 20, duration of diabetes less than 10 years, no vascular disease

Class C Onset of diabetes between ages 10 and 19, duration of diabetes 10–19 years, no vascular disease

Class D Onset of diabetes under age 10, duration of diabetes 20 or more years, vascular disease, including calcification of leg vessels, diabetic retinopathy

Class E Same as Class D, plus calcification of pelvic vessels

Class F Diabetic nephropathy (often Kimmelstiel-Wilson intercapillary nephrosclerosis)

Many of the emergency situations involving diabetes mellitus in pregnancy can be avoided if the patient is seen jointly by the obstetrician and the internist at least every 2 weeks throughout pregnancy Overt diabetics usually require insulin, and as a rule, the requirement is greater during pregnancy Blood glucose determinations should be used for regulation of diet and insulin As general management of the diabetic patient is covered in medical textbooks, we will concern ourselves only with emergency situations in the pregnant woman and the desirable method of delivery Sepsis, toxemia, and hemorrhage are the most common causes of maternal death, and all of these are more common in the pregnant diabetic

MANAGEMENT

Diabetic Coma

The management of the patient with diabetic coma has been well covered by Weil and Shubin (Table 14-1)

Insulin Shock (Hypoglycemic Coma)

If a patient is comatose, and it is not possible to differentiate rapidly between diabetic coma and insulin shock, she should be first treated as though she had a hypoglycemic attack Treatment consists of slowly administering 20–50 ml of 50% glucose in water intravenously If the patient is in insulin shock, she will recover rapidly If she does not, then diabetic acidosis is probably the reason for her coma, and she should be treated as suggested in the foregoing section

Delivery

The patient with any significant degree of diabetes mellitus should be admitted to the hospital at about the 36th week She may have to be admitted earlier if she has toxemia of pregnancy or polyhydramnios, or if her diabetes cannot be controlled on an outpatient basis If a patient is taking insulin for diabetes, it is generally advisable to deliver her before the 38th week In a patient with severe diabetes, delivery may be desirable as early as the 34th week However, other factors must be considered when this decision is being made, and serial urinary estriols, ultrasonography, L/S ratios, and stress tests for the fetus are all useful aids As a general rule, patients should be delivered vaginally, but if this is not practical, cesarean section should be carried out

When the decision has been made to deliver a patient with severe diabetes mellitus, whose cervix is 50% effaced and 1–2 cm dilated, with a presenting part at station 0, the membranes should be ruptured and an oxytocin infusion begun at a rate of 4 mU/min The dosage is increased as necessary according to the plan, outlined in Chapter 9, Intrapartum Emergencies Continuous monitoring of contractions and fetal heart tones is mandatory

If the cervix is thick and closed, and pointing posteriorly, then delivery is unlikely to occur within 12 hours, so elective cesarean section would be the best method for delivery If a patient has significant diabetes mellitus and a decision has been made to deliver her vaginally, it is important that a 12-hour trial of

TABLE 14-1. Therapy of Diabetic Coma

Abnormality	Treatment	Guides
	Insulin	
Hyperglycemia and ketosis	Insulin injection, regular, 100–200 units IV every 2 hours	Until serum glucose is approximately 300 mg/100 ml
	Insulin injection, regular, 10–50 units every 2–4 hours	Until ketosis is completely reversed and patient is able to eat
	Cover insulin with 5% glucose IV (usually in water), or with oral glucose if patient is able to eat	*Urine sugar 0 to 2+ or serum glucose much below 300 mg/100 ml*
	Double dose of intravenous insulin to 400 units or more until serum glucose declines	If serum glucose fails to decline
	Fluids (based on 70 kg body weight)	
Dehydration and acidosis	One liter of sodium bicarbonate-*saline mixture (Add 80 ml of* 7.5% $NaHCO_3$ *[71 mEq Na^+ and* 71 mEq HCO_3] to 1 liter of 0.45% NaCl [77 mEq Na^+ and 77 mEq Cl^-]	Correction of acidosis as indicated *by rises in venous pH and serum* bicarbonate
	One liter of sodium bicarbonate saline mixture as above (or 1 *liter of 0.45% NaCl)*	Preclude undue increase in central venous pressure and *maintain normal output of urine*
	One to three liters of hypotonic solution (a modified Butler's solution is used on our service which contains per liter: 57 mEq Na^+, 50 mEq Cl^-, 25 mEq K^+, 25 mEq lactate, 6 mEq Mg^{++}, and 7mM *phosphate)*	
	5% glucose in water	Futher need for parenteral fluid *therapy on basis of above* indication. Patient unable to eat. Serum glucose levels below 300 mg/100 ml
	Potassium	
Potassium depletion	While the serum potassium is elevated, no potassium is given *intravenously*	Repetitive measures of serum potassium and monitoring of *electrocardiographic changes*
	Usually, 25 *mEq of potassium* chloride is added to 3rd, 4th, and 5th liters of fluid (each already containing 25 mEq of potassium). Rate of potassium *administration should be less than 25 mEq/hour*	
	If additional fluid is given as 5% glucose in water, 25 mEq potassium chloride is added to each liter	

TABLE 14 1 Therapy of Diabetic Coma (continued)

Abnormality	Treatment	Guides
Severe potassium deficiency	Start potassium therapy with first or second liter of fluid More concentrated solutions may be required (100 mEq potassium per liter)	Continuous electrocardiogram and frequent repetitive serum potassium measurements
Circulatory deficit (shock)	Human serum albumin* (5% intravenously	Monitor central venous pressure blood pressure and urine volume
Severe anemia	Whole blood	Increase hemoglobin to level above 10 g/100 ml

*In the series of cases presented plasma was used This has been discontinued because of the danger of serum hepatitis

(From Beigelman PM Grant WJ Severe diabetic ketoacidosis In Weil MH Shubin H (eds) Critical Care Medicine New York Harper & Row 1975)

labor be given If there is no significant progress by this time, then cesarean section should be performed

CARE OF THE NEWBORN

A pediatrician should be present at the delivery of the baby The cord is clamped immediately Blood is obtained for a blood sugar determination every 3 hours for the first 12 hours If the level is less than 30 mg/100 ml glucose should be given slowly in an intravenous dose of 65 mg/kg of body weight in 0 25% N saline intravenously slowly The baby should be kept warm in a heated crib It should be given 4 ml 20% glucose in water through a rubber-tipped medicine dropper every hour for at least 12 hours It should then be started on 5% glucose in milk, followed 12 hours later by a milk formula Vitamin K_1, 1mg, should be given intramuscularly

The baby should usually be kept in oxygen, 30–40% concentration, with 55% humidity at a temperature of 80–85° F It is kept under close observation for evidence of tremors, convulsions, or respiratory difficulties As these babies are prone to develop the respiratory distress syndrome, the nursery staff should report any respiratory difficulty immediately The neonates should also be observed for evidence of congenital anomalies, acid base abnormalities, hyperbilirubinemia, and hypocalcemia

CEREBROVASCULAR ACCIDENTS

Pregnant women have a higher incidence of cerebrovascular accidents than nonpregnant women This may be explained by collagen changes in the blood vessels during pregnancy Subarachnoid hemorrhage from all causes is more common during pregnancy Recurrent subarachnoid hemorrhage or the chance thereof may be an indication for cesarean section It is unwise to allow a patient to bear down in the second stage if she is known to have a congenital aneurysm of the circle of Willis

Hypertension associated with eclamptogenic toxemia, especially in the presence of intravascular coagulation fibrinolysis syndrome and increased intracranial pressure due to 'bearing down' in the second stage of labor, may cause rupture of congenital cerebral aneurysms, arteriovenous malformations, thrombosis of cerebral veins, or bleeding from any weakened cerebral arteries Some of these cerebrovascular accidents could be avoided postpartum if oxytocics were avoided in patients with hypertensive disease in pregnancy A massive intracerebral hemorrhage may occur in a pregnant woman, especially if she is hypertensive (See Chapter 6, Eclamptogenic Toxemia) This condition is managed in essentially the same way in pregnant and nonpregnant women

SUBARACHNOID HEMORRHAGE

This condition occurs most commonly between 25 and 30 years of age and so may be met with in the pregnant woman When cerebral arterial rupture occurs in pregnancy, the mortality is two to three times that in the nonpregnant woman The condition may be detected prior to rupture if a careful physical examination is made Ocular palsies, diplopia, squint, and facial pain due to pressure on the third, fourth, fifth, and sixth cranial nerves may be present Visual loss and a bitemporal field defect signify pressure on the optic chiasma. Pressure on the optic tract produces homonymous hemianopia

Sudden severe headache with nausea and vomiting follows rupture The patient may become semicomatose or even comatose Nuchal rigidity and bilateral positive Babinski signs are present On spinal tap the cerebrospinal fluid is bloody and under increased pressure

Diagnosis

Cerebral angiography is the best diagnostic means All four cerebral vessels should be inspected because several aneurysms may be present, and rupture will result in subarachnoid hemorrhage

Course

With cessation of active hemorrhage, the cerebrospinal fluid gradually clears and the pressure returns to normal in about 3 weeks The mortality from the first hemorrhage is about 35%, an additional 15% of patients die from a subsequent arterial rupture in the first few weeks A second rupture after 6 months is relatively uncommon In general, the prognosis is poor with cerebral aneurysm but better with bleeding from arteriovenous malformations The prognosis is best when no lesions are discovered with four vessel arteriography, probably because the bleeding source was small and possibly had collapsed or thrombosed at a later stage

Management

The patient should be kept at rest, and exertion should be avoided Surgical measures designed to trap or obliterate the aneurysm are gaining in popularity, and results are improving It is difficult to evaluate the results of surgical treatment, however, because patients with the worst prognosis are not candidates for surgery, and the patients who do best after surgery are those whose prog-

nosis would be relatively good in any case Nevertheless, because of the danger of recurrent bleeding, surgery should be tried if an aneurysm can be identified

THYROTOXICOSIS

Thyrotoxicosis is a serious problem for both mother and unborn child, principally because it tends to be overtreated, resulting in the development of a hypothyroid state in the mother and her baby During the first and early second trimester, this may result in fetal maldevelopment, cretinism or goiter All pregnant women with thyrotoxicosis should be hospitalized and management carefully planned in consultation with an internist and surgeon Therapy is *individualized according to the degree of thyrotoxicosis and the duration of* pregnancy The prognosis is generally excellent for mother and fetus if normal thyroid function can be achieved promptly and then maintained either by medical or surgical measures or a combination of both The most useful laboratory tests are those of total T_4 and T_3 resin uptake

THYROID CRISIS

This is an acute exacerbation of all symptoms of thyrotoxicosis, accompanied by fever, cardiac arrythmia, and extreme tachycardia Heart failure may occur, and the outcome may be fatal Nausea vomiting, diarrhea, hepatomegaly, jaundice, tremors, and delirium are all part of the clinical picture The thyroid storm may be precipitated by infections, unusual emotional stress, or labor Most often, however, it is precipitated by surgery in a patient who has not been adequately prepared medically

Management

The management is as follows

1 Give sodium iodide, 1 g/day intravenously, to inhibit the release of thyroid hormones
2 Give oxygen
3 Give propylthiouracil, 1200 mg/day orally in divided doses
4 Give reserpine 2 5 mg intramuscularly every 6 hours, or propranolol 1–2 mg intravenously then 20 mg every 6 hours orally to control the maternal heart rate with direct monitoring by ECG
5 Hydrocortisone, 300 mg intravenously every 24 hours, is useful, and digitalis and diuretics are used in conventional doses

Intravenous fluids, 6000–7000 ml may be necessary to combat dehydration, and replacement of electrolytes is an essential supportive measure Hypothermia may be necessary for the hyperpyrexia and phenothiazines have been suggested for use in the presence of severe hyperpyrexia

PARATHYROID DYSFUNCTION

The development of mild hyperparathyroidism is a normal concomitant of pregnancy Severe chronic hyperparathyroidism causing osteitis fibrosa cystica is rare during pregnancy, except in patients who have long standing renal dis-

ease The most serious problems relating to parathyroidism in pregnancy are muscle cramps and hypoparathyroid tetany

HYPOPARATHYROID TETANY

This condition is associated with a deficiency of calcium or an excess of phosphate, or lack of vitamin D and parathormone In hypoparathyroidism, hypocalcemia is observed during pregnancy as a dilutional phenomenon The requirements for vitamin D and calcium are greater in the pregnant than in the nonpregnant woman Tetany may occur in late pregnancy if calcium supplements have not been taken, it may be precipitated by hyperventilation during labor, it may follow infection, or it may be caused by the hypocalcemia that sometimes occurs during lactation

Tetany from parathyroid deficiency is characterized by low serum calcium, high serum phosphorus, normal alkaline phosphatase, and absence of urinary calcium In contrast, in alkalotic tetany due to hyperventilation, the most common form of tetany, the alkaline urine contains normal amounts of calcium

Management

Treatment consists of slowly administering 10% calcium gluconate, 1–3 g intravenously This dosage may be repeated in 6 hours

Maintenance therapy consists of the administration of vitamin D_2 (calciferol) 50,000–150,000 IU/day orally This replaces parathyroid hormone, raising serum calcium to normal, lowering tubular reabsorption of phosphate, and thereby reducing the serum phosphorus level Symptoms are relieved slowly, since vitamin D acts slowly but persistently Because the most common cause of hypoparathyroidism is the accidental removal of or damage to the parathyroid glands at the time of thyroidectomy, *hypoparathyroid tetany will tend to be worse in the post thyroidectomy patient who becomes pregnant*

ULCERATIVE COLITIS

Ulcerative colitis has little effect on fertility, but pregnancy may have a profound effect on this disease under certain circumstances When pregnancy occurs while the colitis is inactive, an exacerbation is unlikely However, when conception coincides with active ulcerative colitis, 50–75% of the patients will suffer a severe relapse during pregnancy or in the puerperium When ulcerative colitis has its onset during pregnancy, more than 50% of the patients will have a severe attack, and some will die When colitis has its onset during the puerperium, most patients will have a protracted course Pregnancy almost never exerts a favorable effect on the course of ulcerative colitis

Most authorities consider that therapeutic abortion is medically justified in cases of acute, fulminating, treatment-resistant ulcerative colitis that is exacerbated by pregnancy

In an acute fulminating episode, the patient suddenly develops violent diarrhea, vomiting, cramps, high fever, and profound toxemia The stools are watery and contain foul fecal matter mixed with mucus, pus, and often gross blood.

The diagnosis is confirmed by proctosigmoidoscopy or colonoscopy

Management

There is no specific treatment for ulcerative colitis Strict bedrest and fluid and electrolyte replacement are the prime features of management When anemia is severe, transfusion with compatible blood is indicated

Diphenoxylate (Lomotil), 2 5–5 mg orally four times daily, or a combination of deodorized opium tincture, 0 6–1 5 ml, and belladonna tincture, 0 6 ml orally every 4 hours are effective in reducing cramps and stool frequency Anticholinergic drugs and opiates should be given at regular intervals rather than according to the number of stools In severe cases none of these agents are very effective in controlling the number of bowel movements Severely ill, toxic patients with a high fever are in danger of perforation of the bowel and should be given antibiotics effective against the intestinal flora The placement of a gastrointestinal tube is useful for the administration of antibiotics, and suction can be used as necessary In seriously ill patients, it is worth while to give hydrocortisone, 100 mg intravenously, three times daily Although not curative, glucocorticoids induce remission in over 60% of patients

Surgery is indicated for ulcerative colitis under the following circumstances 1) failure to respond to medical measures, 2) progressive abdominal distention with innumerable stools and anemia, 3) massive hemorrhage, 4) colonic obstruction, 5) suspected carcinoma

The most effective surgical measure is total proctocolectomy with establishment of a permanent ileostomy Because of the excessive loss from an ileostomy, careful supervision of the patient in the hospital is essential before she is allowed to go home

SYSTEMIC LUPUS ERYTHEMATOSUS

Systemic lupus erythematosus is characteristically a disease of women and develops most frequently during the childbearing years The maternal mortality is approximately 20% and the perinatal mortality is approximately 30% in acute disseminated lupus erythematosus The mortality in chronic systemic lupus erythematosus depends upon the duration and severity of the disease

Pregnancy does not consistently influence the course of this disorder In over 50% of the patients with lupus erythematosus, the disease remains unchanged, and in a few cases it appears to improve during pregnancy Neither the disease itself nor its treatment with corticosteroids commonly reduces the fertility of the patient Spontaneous abortion usually before the 14th week, occurs in about 20% of the patients with acute lupus erythematosus The incidence of premature labor and toxemia of pregnancy is also increased

Cardiac insufficiency is often a critical problem, but progressive renal failure is the most frequent contributory cause of death in pregnant women with lupus erythematosus

Treatment

All patients must be admitted to the hospital for treatment of acute lupus erythematosus Prednisone, 30–50 mg orally, in four divided doses daily, may be required for the treatment of an acute attack After improvement has oc-

curred, a maintenance dose of about 10 mg/day may be used for a prolonged period during pregnancy and the puerperium Patients should avoid exertion and exposure to ultraviolet light Pigmented, emollient, cosmetic lotions that are opaque to ultraviolet light may be applied to the face lesions

Pregnancy rarely exacerbates lupus erythematosus so severely that therapeutic abortion is justified Cesarean section should be used for delivery only if an obstetric indication is present

GLOMERULONEPHRITIS

An initial attack of acute glomerulonephritis is rare during pregnancy, most obstetric problems relating to glomerulonephritis involve transitional, chronic forms of the disease There is no evidence that pregnancy aggravates glomerulonephritis Nephritis causes hypertension, predisposes to eclamptogenic toxemia, and contributes to an increase in perinatal mortality and morbidity

The treatment of glomerulonephritis in pregnancy is the same as that in the nonpregnant patient Antibiotics are ineffective, and glucocorticoids may be harmful

The disease may be an indication for cesarean section when placental dysmaturity or eclamptogenic toxemia occurs

If azotemia is present in the patient with acute glomerulonephritis, dietary protein is restricted Sodium and water intake are restricted only when edema or severe hypertension is present Diuretics such as furosemide are sometimes helpful in the management of marked edema or severe hypertension Hypertensive encephalopathy may require parenteral therapy with antihypertensive agents, such as methyldopa (Aldomet) in a dosage of 250–500 mg four times daily

EXANTHEMATOUS DISEASES

This group of diseases is caused by viruses that invariably gain access to the fetus by crossing the placental ' barrier " The effect of these organisms on pregnancy and the fetus depends upon the virulence of the disease, the mother's resistance to it, and the stage of fetal development at which infection occurs

Fetal immunity depends largely on maternal active immunity (*e.g*, active immunity from smallpox) or passive immunity (*e g*, from measles vaccination administration) Following a viral placentitis, fetal death *in utero* may occur, with abortion or premature delivery The effects of the exanthematous diseases have been analyzed by Benson (Table 14-2)

Exposure to Rubella in Pregnancy

In some respects this is a medical emergency, inasmuch as the problem is an extremely difficult one The pregnant woman who is exposed to rubella in the first trimester has a 15–20% chance of having an abnormal baby If a patient is exposed to a proven or strongly suspected case of rubella, Schwartz (1973) suggests that the following steps be taken

TABLE 14-2. Exanthematous Diseases in Pregnancy

Disease	Effect of disease: On pregnancy	On offspring
Variola (smallpox)	*Abortion premature delivery* frequent postpartal hemorrhage maternal mortality increased	*May be born with pocks*
Rubeola (measles)	Abortion premature labor if disease is severe	May be born with rash
Varicella (chickenpox)	Severe disseminated epidemic type may be fatal to mother as result of necrotizing angiitis	Virulent infection may cause fetal death in utero neonate may be born with pocks
Rubella (German measles)	Occasional early abortion	Congenital anomalies if disease occurs during first trimester

(Benson RC Handbook of Obstetrics & Gynecology Los Altos CA Lange Medical Publications 1974)

1. Establish the susceptibility of the patient (10–20% of women are susceptible) by measuring rubella antibodies A history of prior infection is not sufficient If immunity is demonstrated, the patient can be reassured and nothing further need be done Titers indicative of immunity vary from laboratory to laboratory, but usually *if greater than 1 10 they indicate immunity*
2. If the patient is susceptible, she should be observed for development of clinical disease, as well as for a serologic response A repeat rubella antibody titer should be done 2–3 weeks after the initial one A rising titer is indicative of infection, even in the absence of clinical signs
3. With *the ready availability of abortion, many patients will elect to have this* procedure
4. The giving of gamma globulin may obscure the clinical disease without preventing teratogenic effects However, its use should be considered as an alternative for a patient who will not accept abortion

POLIOMYELITIS

This condition exerts an unfavorable effect on pregnancy Poliomyelitis is now *more rare than it was formerly*, but the disease increases the risk of abortion and fetal loss Moreover, the fetus may contract poliomyelitis in its passage through the birth canal Bulbar poliomyelitis, most often observed in late pregnancy, carries the most serious prognosis for both mother and fetus, and the danger of losing either is greater in the third trimester

Paralysis of the intercostal muscles, the diaphragm, and the abdominal *muscles, together with the* enlarged uterus, reduces maternal respiratory exchange when the patient is in a tank respirator Thus, early delivery, often by cesarean section, enhances mechanical ventilation by the tank and contributes to the survival of the mother and infant Emergency treatment in acute bulbar

poliomyelitis includes early tracheostomy, endotracheal suction, administration of oxygen and helium under positive pressure, and antibiotics Gamma globulin should be given to the newborn to protect it against poliomyelitis

INCOMPATIBLE BLOOD TRANSFUSION REACTION

A hemolytic transfusion reaction occurs when an individual receives blood from a donor containing red blood cells of a type to which the recipient is sensitized or has naturally occurring antibodies As pointed out by Schwarz (1973), nearly all such reactions are the result of a clerical error. Lysed donor cells release their hemoglobin, which is deposited as acid hematin in the renal tubules and can cause acute tubular necrosis The release of the lysed red cell contents can activate the coagulation mechanism and result in disseminated intravascular coagulation, with depletion of fibrinogen as well as the deposition of microthrombi in vital organs The released red cell contents may also activate plasminogen to plasmin, with consequent degradation of fibrin to fibrin split products with their heparinlike activity Allergic manifestations may accompany the transfusion reaction Major hemolytic transfusion reactions require prompt treatment

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Surgical Emergencies in Obstetrics

Denis Cavanagh

Chapter 15

The slings and arrows of outrageous fortune"
William Shakespeare (1564–1616)
Hamlet Act III Scene 1

When immediate surgical intervention is necessary, the presence of a pregnancy may prompt special measures, but the surgical need of the mother must be the primary consideration However, the possible effect of the surgical treatment on the pregnancy itself must also be considered Also, many antibiotics used to combat life threatening infections during pregnancy have not been approved for use in pregnancy by the Food and Drug Administration and so the benefit to the mother must be balanced against possible damage to the fetus

Among 17,000 pregnancies at the Beverly Hospital, MA, Garry (1957) reported a series of 32 laparotomies carried out during pregnancy Twenty seven of these women carried their babies to term Three women had hysterectomies, and two delivered five premature infants who died within 24 hours of birth Shnider and Webster (1965) reported that over a 49 month period, 147 of 9073 women who were delivered had some type of surgery done during their pregnancy Their second series, compiled in a cooperative study of 17 hospitals, included 50 appendectomies in the course of 60 912 pregnancies Premature labor and delivery were due chiefly to the indication for surgery rather than the procedure itself although, as would be expected, women who had surgery for incompetent cervical os demonstrated a high incidence of premature labor (28%) and a high perinatal mortality (33 3%) The type of anesthesia used did not appear to affect the outcome in terms of prematurity, but whether anesthesia exerts a deleterious effect on the fetus awaits the findings of further research With the recent discovery in animal studies that some anesthetic agents are teratogenic and the fact that the uterus is less irritable in the second trimester, elective surgical procedures during pregnancy should be postponed until the second trimester

APPENDICITIS

This condition complicates one pregnancy in approximately 1200 Although no more common among pregnant women than in the general population, this acute surgical condition carries with it the difficulty of diagnosis and the increased danger of generalized peritonitis When diagnosed, or even strongly suspected surgery is immediately indicated, regardless of the stage of the pregnancy Uterine irritability may be diminished by the use of progesterone, but careful manipulation can prevent many of the obstetric complications Peritonitis, when it does develop, can produce uterine irritability, as it occurs most often in the first two trimesters, abortion and premature birth frequently result According to Douglas and Stromme (1965), when a patient is operated upon before perforation occurs, abortion or premature labor is unlikely

Diagnosis

1. Pain is classically of the periumbilical type, then migrates to the right lower quadrant
2. Anorexia, nausea and vomiting are common in pregnancy, so a high index of suspicion is required for the diagnosis of appendicitis to be made
3. Tenderness is usually localized to the right lower quadrant, but the position changes as pregnancy advances Moreover, the obstetrician must be aware that maximum tenderness rarely coincides with McBurney's point (Fig 15-1)
4. Mild leukocytosis is normal in pregnancy and may be confusing Usually there is a shift to the left in the patient with appendicitis Urinalysis is important, since pyelonephritis must be ruled out, but there may be white cells in the urine because ureteral proximity to an acutely inflamed retrocecal appendix results in this

Management

Immediate surgery is imperative Even though exploratory laparotomy does not reveal an acute appendix in about 50% of the patients, the danger of surgery is much less than the danger of possible rupture and peritonitis If peritonitis is allowed to develop the result will be a significantly worse prognosis for both the mother and her baby In the operating room, and in the postoperative period, several points should be kept in mind

1. Anesthesia may be regional or general, as long as hypoxia and hypotension are avoided Halothane anesthesia will relax the uterus and may help to prevent premature labor
2. Use a right paramedian incision This will give access to the appendix at any site and will permit adequate exploration of the upper and lower abdomen if the diagnosis is wrong A McBurney incision should *not* be used in women, whether pregnant or nonpregnant, because if the diagnosis is wrong it does not permit adequate exploration of the pelvis and abdomen
3. Inject ampicillin, 1 g every 4 hours intravenously, for the first 24 hours after operation, thereafter 1 g every 6 hours should be given for 7 days If the appendix is ruptured chloramphenicol (Chloromycetin) 1 g every 8 hours or clindamycin (Cleocin) 600 mg every 6 hours should be given intravenously for 7 days

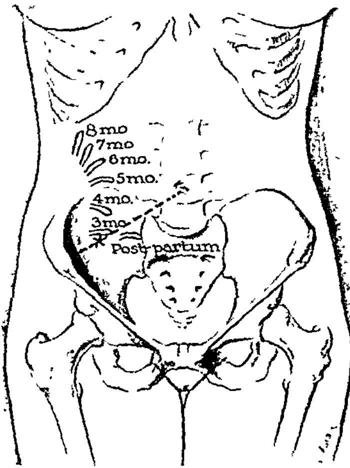


FIG. 15-1. Diagrammatic representation of probable location of appendix at different months of pregnancy and the puerperium X marks McBurney's point. (Baer JL et al JAMA 98 1359, Copyright 1932, American Medical Association)

4. As in all situations where peritonitis and uterine irritability are present, the use of progesterone in oil, 100 mg intramuscularly, as an initial dose, and hydroxyprogesterone (Delalutin), 250 mg daily for 7 days, may be worth while.
5. In a patient near term, cesarean section should generally be reserved for obstetric indications In the presence of perforation or abscess formation, if cesarean section is indicated, hysterectomy should also be considered.

MECKEL'S DIVERTICULITIS

This small bowel diverticulum is about 2 in. long, arises about 2 feet from the ileocecal valve, and is found in about 2% of Caucasians. (The eponym is unfortunate, as the diverticulum was clearly described by John Hunter long before it was reported by Meckel)

Everything that has been said about acute appendicitis may be applied to Meckel's diverticulitis In fact, the diagnosis is usually made only after the abdomen has been opened following a diagnosis of acute appendicitis Meckel's diverticulitis treated surgically in a pregnant woman was reported by Walser *et al*, the patient was delivered of a term infant

As with all other intraabdominal conditions the tendency for a patient to go into labor depends primarily on the degree of peritonitis When the peritoneum over the uterus becomes involved, the result is probably increased uterine irritability It is postulated that there is then a local disturbance of progesterone balance, which in some way triggers the release of endogenous oxytocin from the neurohypophysis Nevertheless, it seems clear that the welfare of both mother and fetus is best served by early abdominal exploration in any patient with a 'surgical abdomen'

ACUTE PELVIC INFLAMMATORY DISEASE

Gonorrhea in pregnancy is not uncommon but is usually asymptomatic The pregnant woman can serve as a source of infection for sexual contacts and for her baby's eyes She should therefore be treated despite her lack of symptoms Acute pelvic inflammatory disease in pregnancy is rare

The diagnosis is usually made at laparotomy in a patient in whom a diagnosis of acute appendicitis has been made

Management

The treatment is as in the nonpregnant state If a tuboovarian abscess is present, the affected tube and ovary should be removed As in acute appendicitis, ampicillin, 1 g every 4 hours intravenously, with chloramphenicol, 1 g every 8 hours or clindamycin 600 mg every 6 hours intravenously, should be given for 7 days if abscess formation has occurred

Cultures are taken 14 days after treatment and repeated as necessary until the patient is cured Smears are not adequate for diagnosis in women, so cultures must be obtained Cultures of endocervix and anal canal are taken These are plated directly onto Thayer Martin medium and placed in a suitable incubator jar Alternatively, these may be placed on special transport medium and sent to the laboratory

OVARIAN TUMORS

Ovarian tumors and enlargements, benign and malignant, are not uncommon in the childbearing years The most common ovarian tumor found in early pregnancy is the corpus luteum of pregnancy True neoplastic tumors are rare, with the exception of the benign cystic teratoma ("dermoid"), which accounts for 25% of all ovarian tumors found during pregnancy Frequently diagnosed in the first trimester of pregnancy at the initial pelvic examination, they may grow insidiously and become quite large in the second and third trimester because a second pelvic examination is not usually performed until the 36th week of pregnancy Ovarian tumors are subject to many accidents, such as hemorrhage, torsion, and rupture, and therefore they can precipitate the need for immediate

surgery during any stage of pregnancy Asymptomatic tumors may be observed in the first trimester, and the operation should be postponed until 16–20 weeks if possible, because at that time the uterus is less irritable and the danger of teratogenicity is over

Diagnosis

Tumors, cystic or solid, can be readily missed during pregnancy, but usually will produce few symptoms On occasion, the consequences may be extremely serious Pedunculated, cystic ovarian growths will usually rise out of the pelvis and will rarely prevent vaginal delivery On the other hand, they may undergo torsion at any stage of gestation and require immediate surgical intervention While counterrotation may correct the torsion, thrombosis and necrosis have usually occurred by the time surgery is performed, and oophorectomy is usually necessary If torsion occurs early in pregnancy, before the placenta is well established, uterine contractions may follow In this situation progesterone in the same dosage as that used following appendectomy may be effective Expulsive contractions may also occur when a laparotomy is performed in the later months, but the placental production of progesterone, and the greater refractoriness of the uterine musculature at these stages of pregnancy, reduces this possibility as compared with the first trimester Most important is gentleness of manipulation at surgery and the avoidance of peritonitis from spillage

Solid tumors tend to gravitate into the pelvic cavity and more frequently interfere with vaginal delivery Unless torsion occurs they will have little influence on the prematurity rate Solid tumors may exist throughout pregnancy and may not be discovered until they interfere with descent of the fetus in labor or until they are felt in the relatively empty pelvis during the puerperium Of far greater significance is the solid tumor discovered during gestation itself On the basis of probability, the tumor will be the benign solid type or a benign cystic teratoma, but the possibility of malignancy must be considered

Torsion is the *most frequent complication of ovarian cysts in pregnancy, occurring in about 12% of patients with ovarian tumors* and is often accompanied by rupture Torsion occurs most often in the first trimester, but it can occur at any time The clinical picture has several characteristics

1. Unilateral, often intermittent, colicky pain at the onset later becomes constant and more severe as gangrenous changes occur The pain may be either in the upper or lower abdomen Often a cyst is compressed behind the uterus in the cul-de sac and posterior pelvis, causing backache and pain radiating down the legs
2. The presence of an adnexal mass can usually be detected, unless muscle guarding prevents adequate examination Palpation of an adnexal mass becomes increasingly difficult as pregnancy progresses Pelvic examination under anesthesia is prudent when the patient is uncooperative or when there is any doubt in the examiner's mind about the pelvic findings
3. Signs of peritonitis and fever occur late, and indicate gangrenous change or rupture When an ovarian cyst ruptures tenderness, rebound, and muscle guarding are marked, and operation is urgently required In addition to the immediate peritonitis these patients develop marked adhesions which may result in recurrent intestinal obstruction
4. Laboratory studies are not helpful in the early stages, but in the more advanced stages, leukocytosis with a *shift to the left* may be seen The white

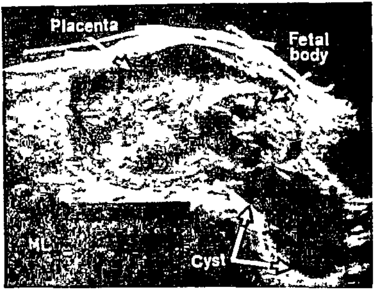
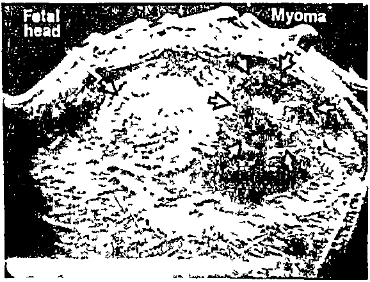


FIG 15 2 Sonogram, longitudinal section at midline showing a large cystic mass occupying the pelvis Scattered areas of increased density within the cyst suggest that it is benign cystic teratoma (Courtesy of Dr R. E Woods)

FIG 15 3 Sonogram, transverse section at fundus of uterus, showing fetal head and a large, sonolucent myoma (Courtesy of Dr R E. Woods)



blood count may range from 10,000 to 14,000/cu mm in normal pregnancy

5. A single flat x-ray film of the abdomen may be helpful if it reveals a diagnostic pattern of calcification or a tooth in a benign cystic teratoma
6. Sonar examination may identify the nature of the pelvic mass (Fig 15-2, 15-3)

Management

In the presence of torsion or rupture of an ovarian cyst, prompt surgical intervention is the only treatment Only this action can avoid the serious problem of peritonitis Peritonitis from a ruptured benign cystic teratoma is especially serious because of the serious chemical peritoneal irritation from the cyst contents In any event, with peritonitis there is also a significant risk of abortion or premature labor When a twisted cyst or tubal mass is removed, it should *not* be untwisted prior to removal, for this may release a thrombus in the venous system

When a suspicion of malignancy arises, surgical intervention becomes imperative If the suspicion becomes certainty at the time of surgery, further immediate treatment is that indicated by the nature of the pelvic malignancy If the fetus is viable at the time of this surgery, cesarean section should be done just before any hysterectomy and adnexal extirpation

Booth (1963) reported on 50 women who had ovarian tumors during pregnancy One of them aborted at 11 weeks, and 46 others delivered at term The premature rate in this series was an unremarkable 8%

Peterson *et al* (1955) found an increased incidence of spontaneous abortion and premature labor when pregnancy was associated with an ovarian tumor They believed that the effect was mainly that of the accompanying complications and the emergency surgery that followed

Results

Garry (1957) reported three cases of torsion of ovarian cysts among 32 laparotomies that were done for ovarian growths One of these women was delivered of a premature, live child one day after oophorectomy at 6 months, but the child failed to survive

Ottaway (1963) reported two cases of ruptured hemorrhagic ovarian cysts during pregnancy, one woman was subsequently delivered at term, and the other was delivered, at the 38th week, of a 2455 g infant after induction of labor for Rh sensitivity Nineteen patients with ovarian tumors were listed by McCorriston (1963), and no premature labors ensued An excellent review of the complications in patients with benign cystic teratomas was made by Pantoja *et al* in 1975

CERVICAL OPERATIONS

Cone Biopsy

The advent of colposcopy has much reduced the need for cold knife cone biopsy in the pregnant patient with a positive Papanicolaou smear

Schulman and Ferguson found no increase in abortion or premature labor

among 41 patients who became pregnant following cold knife conization of the cervix, as compared with a control group, although two patients had dystocia and severe cervical lacerations presumably from post-conization cervical fibrosis

When cold-knife conization is performed during pregnancy, however, the tendency to both abortion and premature labor is increased Therefore, in the presence of a positive Papanicolaou smear, with a punch biopsy diagnosis of carcinoma *in situ* or less in the third trimester of pregnancy, diagnostic conization of the cervix is best postponed until the 36th week of pregnancy Consultation with a skilled colposcopist should be sought, because colposcopically directed biopsies are almost as accurate as cone biopsy in establishing the definitive diagnosis

Needless to say, when invasive carcinoma of the cervix is strongly suspected, cone biopsy should be done at any time during pregnancy in the interest of the mother In a study of 50 conizations during pregnancy, Ferguson and Brown found that the operation posed little threat to the mother but in eight cases affected the fetus (five of the fetal deaths could not be directly attributed to the conization procedure) They felt that vaginal delivery in the face of invasive cervical carcinoma was not advisable but that it was not contraindicated in cases of carcinoma *in situ* In any event, each case of suspected cervical carcinoma must be individually judged as to the stage of pregnancy when diagnosed and as to the advantages and disadvantages of immediate surgical intervention for diagnosis and treatment

INCOMPETENT CERVICAL OS

Although intervention may not be necessary in all cases, it should be appreciated that in approximately 15% of obstetric patients with this problem, there is a tendency for the uterine cervix to dilate in the course of the growing pregnancy The cervical weakness may be due to prior trauma (as from a delivery or dilatation and curettage) or to a congenital weakness of the cervix If these patients deliver prematurely, they usually do so in the second or third trimester

The typical clinical picture is of bulging membranes with spontaneous rupture, followed shortly thereafter by expulsion of the fetus There are usually no uterine contractions The patient often presents with a complaint of profuse, watery discharge, spotting, or pelvic pressure

Diagnosis

The diagnosis should be made and treatment by operation undertaken if a patient has a past history of abortion or premature delivery On speculum examination, cervical dilation of 2 cm or more, with effacement and perhaps bulging membranes, may be seen (Fig 15-4) If contractions are present, they are usually painless

Management

Bed rest is advisable to observe the patient for the possibility of labor The patient should be placed in the Trendelenburg position to reduce the effect of the fluid wedge on the internal os The use of ritodrine, orciprenaline, etc,

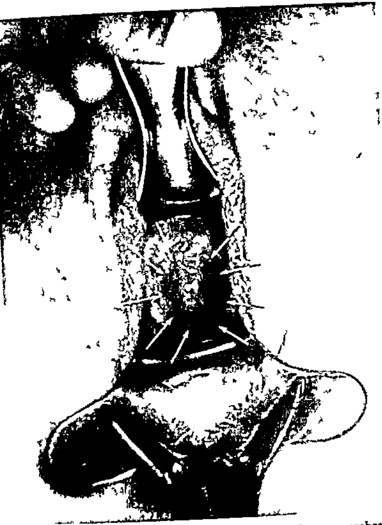


FIG 15-4 Incompetent cervical os with bulging membranes (Courtesy of Dr Hubert A. Ritter)

should be considered to inhibit labor (see Ch 9 Intrapartum Emergencies) Preparations should be made for cerclage operation using the Shirodkar (Fig 15 5 to 15 8), Wurm (Fig 15 9) or Ritter (Fig 15 10) procedures General anesthesia with a uterine relaxing agent such as halothane may be helpful

The results of the operation vary according to the stage at which the operation is performed and the type of procedure followed Recently Ritter Ritter, and Callaghan reported a success rate in the region of 95% (1976) with their operation

ECTOPIC PREGNANCY

It is not too uncommon to be confronted with a woman in acute shock with the classic symptoms of a ruptured tubal pregnancy and to have instituted corrective surgery only to find that she has a coexistent intrauterine pregnancy

FIG 15-5. Shirodkar-Barter operation for incompetent cervix. A. After transverse incision of vaginal mucosa, bladder is displaced upward by scissor dissection. B and C. Bladder has been dissected upward to a level just above the internal os. (Eastman NJ, Hellman L [eds]: Williams Obstetrics, 12 ed. New York, Appleton-Century-Crofts, 1961, p 550)

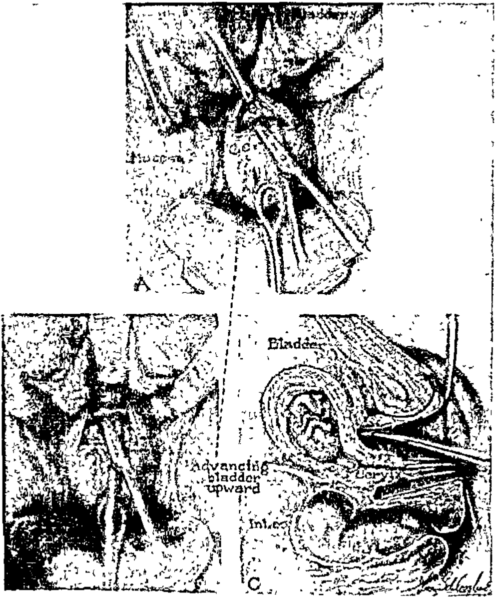
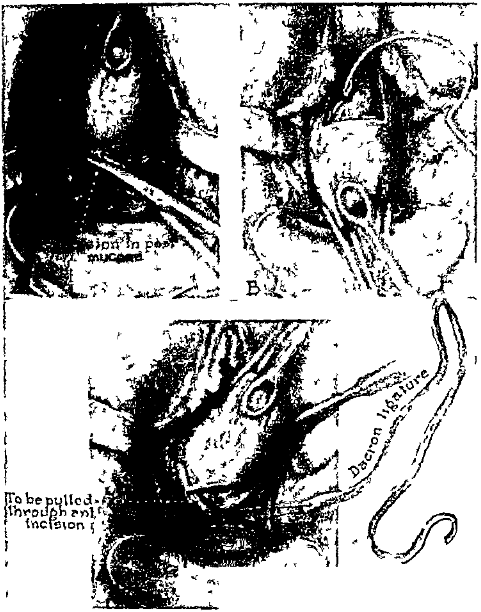


FIG 15 6 Shirodkar-Barter operation (continued) A. Incision in posterior mucosa corresponding to level of anterior incision B Aneurysm needle is about to be passed through anterior incision, under vaginal mucosa and out through posterior incision C. A Dacron ligature is attached to needle (Eastman NJ, Hellman L [eds] Williams Obstetrics, 12 ed New York, Appleton Century-Crofts, 1961, p 55)



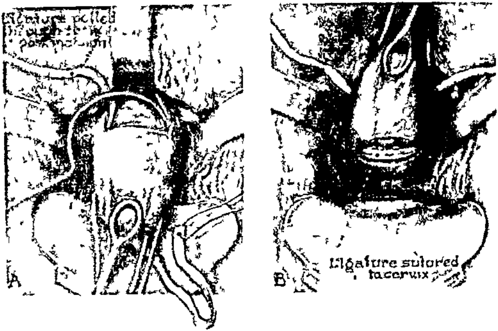


FIG 15 7 Shirodkar Barter operation (continued) A After the ligature has been pulled through the incisions on patient s right the same procedure is carried out on left side B Dacron ligature is anchored to posterior cervix by silk ligature to prevent its slipping over the posterior lip of cervix (Eastman NJ Hellman L [eds] Williams Obstetrics 12th ed. New York Appleton-Century-Crofts 1961 p 552)

As this situation occurs usually in the early months of pregnancy, concomitant oophorectomy may lead to uterine contractions and abortion so only salpingec tomy or salpingostomy should be performed Ectopic pregnancy in the later months is rare it is usually abdominal rather than tubal and is rarely associated with an intrauterine gestation As already pointed out (Ch 7, Hemorrhage in Early Pregnancy) culdocentesis is a very useful diagnostic tool for detecting intraperitoneal hemorrhage and should be employed at any time that ruptured ectopic pregnancy is suspected If ruptured ectopic pregnancy is suspected do not delay surgery to await blood replacement Open the abdomen through a longitudinal incision and clamp the bleeding area

Laparoscopy or culdoscopy should be carried out when an unruptured ectopic pregnancy is suspected and the uterus is not too large to be a contraindication to these procedures

INTESTINAL OBSTRUCTION

Approximately once in 6000 pregnancies intestinal obstruction occurs About 50% happen in the second trimester Obstruction is usually due to adhesions that are the result of a previous appendectomy myomectomy, uterine suspen

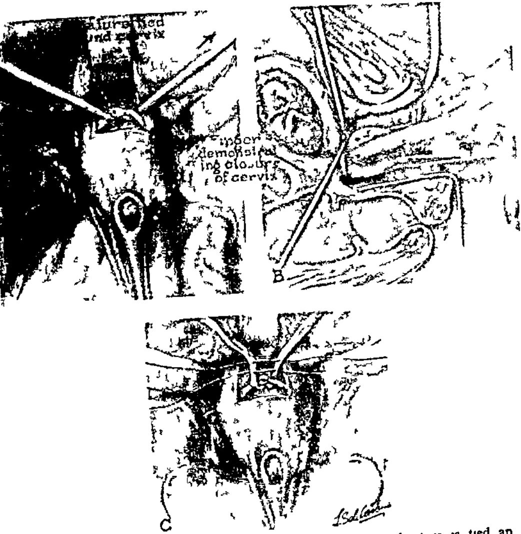


FIG 15 8 Shirodkar Barter operation (continued) A After ligature is tied anteriorly it is anchored by a silk suture B Testing size of internal os it should just admit fingertip C Reinforcing ligature before cutting (Eastman NJ Hellman L [eds] Williams Obstetrics 12th ed New York Appleton-Century-Crofts 1961 p 553)

sion or adnexectomy but it may follow any abdominal operation In an unscarred abdomen it may be due to an incarcerated hernia (internal or external) volvulus or intussusception Diagnosis of intestinal obstruction is difficult because of the enlarging uterus and displacement of the viscera This emergency requires immediate surgery regardless of the stage of pregnancy

Pre and postoperative nasogastric suction and adequate fluid and electrolyte replacement are essential supportive measures An elevated leukocyte count with a shift to the left and a radiographic bowel pattern suggestive of a closed loop obstruction call for immediate surgical intervention Intensive antibiotic therapy is indicated if sepsis or gangrenous bowel is found

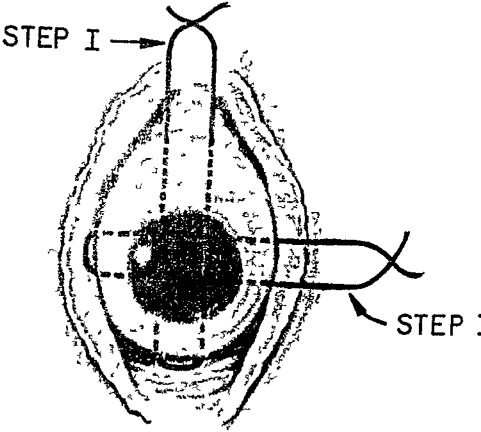


FIG 15 9 Suturing of cervix in Wurm procedure (Hefner et al. Obstet Gynecol 18 616 1961)

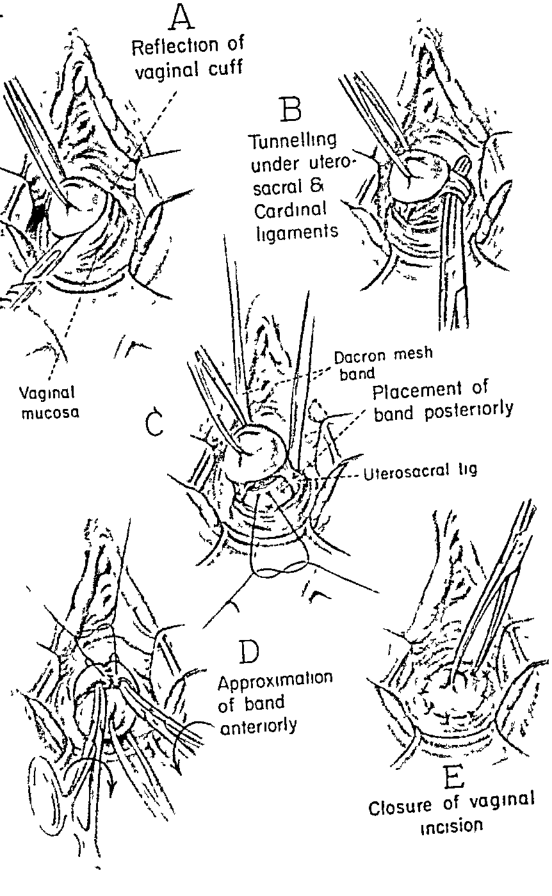
PEPTIC ULCER

In a pregnant woman with a peptic ulcer perforation or hemorrhage may result in a life threatening emergency calling for immediate attention

PERFORATION

Although it rarely does so a ruptured peptic ulcer may initiate the onset of labor by stimulating the serosal surface of the uterus The initial chemical peritonitis is followed by secondary bacterial contamination Surgery should

FIG 15 10 A Circumscribing incision of cervix through mucosa and submucosal fascia B Reflection of vaginal cuff and fascia exposing uterosacral ligaments with development of tunnels C Placement of band posteriorly and through the tunnels. D Approximation of band anteriorly with inversion of free ends E. Closure of cervical incision (Ritter HA Ritter PJ Obstet Gynecol 17 342–345 1961)


A
Reflection of vaginal cuff
B
Tunnelling under utero-sacral & Cardinal ligaments
Vaginal mucosa
Dacron mesh band
Placement of band posteriorly
C
Uterosacral lig
D
Approximation of band anteriorly
E
Closure of vaginal incision

be immediate, regardless of the state or stage of pregnancy Some very large perforated ulcers have been reported during pregnancy

HEMORRHAGE

Operation for a hemorrhaging peptic ulcer is not usually required unless massive hematemasis or melena is present McCorriston reported three cases of women with active duodenal ulcers or duodenitis in pregnancy, none of them delivered prematurely An additional patient with a bleeding duodenal ulcer developed cardiac arrest during surgery Although she recovered following open chest cardiac massage, her baby was stillborn

RENAL EMERGENCIES

PYELONEPHRITIS

Kidney inflammation is not a surgical emergency, but it is sometimes mistaken for acute appendicitis or other causes of acute abdomen

The condition may occur at any stage during pregnancy but is most common in the second trimester The patient suffers anorexia, nausea, and vomiting Costovertebral angle tenderness is present often bilaterally but is more commonly present on the right side Laboratory evidence of infection is the finding of clumps of white cells in a clean catch or catheter specimen of urine In addition, a patient frequently has a leukocytosis with a shift to the left

These patients often have temperatures ranging up to 105° F and should always be considered to be very ill The possibility of septic shock is present in these patients This aspect has been discussed in Chapter 3, Shock

Management

Treatment consists of bedrest, hydration, and antibiotic therapy If possible culture and antibiotic sensitivity studies should be obtained before antibiotic treatment is begun Ampicillin, 1–2 g every 4 hours *intravenously*, will usually control the infection within 48 hours Thereafter the dosage can be reduced to 500 mg every 6 hours The course should be continued for at least 10 days If the patient is not improving and the sensitivity studies show the organism is resistant to ampicillin, this drug should be discontinued and a suitable antibiotic prescribed

If the patient does not respond rapidly have her lie on the opposite side from the kidney that appears to be most markedly infected This will often result in better drainage of the infected kidney Although the disease is characteristically bilateral, the right side is usually more infected than the other

In a patient near term, labor should be induced Following delivery drainage of the calyces is more readily achieved

RENAL COLIC

Ureteral stones are more common during pregnancy than in the nonpregnant state because of the hypercalciuria of pregnancy, the dilatation of the urinary

tract, and the stasis resulting from pressure of the gravid uterus on the pelvic brim Stones up to 0 5 cm will usually pass, but larger stones become impacted The extreme nature of the pain and its location in the back and/or flank make the diagnosis fairly easy A history of previous episodes of renal colic, the presence of hematuria, and a visible stone on x ray or intravenous pyelogram are diagnostic If the stone is not passed, it may be dislodged by retrograde catheterization or even removed transurethrally If this is unsuccessful, progressive hydronephrosis develops so the stone must be removed by extraperitoneal ureterolithotomy

CHOLECYSTITIS

Patients with symptoms of cholecystitis are frequently obese and often have a strong history suggestive of gallbladder disease The location of the pain in the right upper quadrant and a positive Murphy's sign, visualization of stones on x-ray films, or the detection of hyperbilirubinemia suggest the diagnosis Unless essential for immediate treatment a cholangiogram is not indicated because of the radiation to the fetus

Symptomatic relief may be obtained with meperidine (Demerol), 100 mg, and atropine, 0 4 mg, subcutaneously

Gallbladder surgery in pregnant women should be avoided unless obstruction occurs, because it carries a perinatal mortality of approximately 15% In obstruction, however, operation is necessary to prevent rupture of the gall bladder and peritonitis Cholecystectomy, or even cholecystostomy, and the establishment of biliary drainage are essential

PANCREATITIS

This condition is extremely rare in pregnancy The symptoms are vomiting and severe, boring, epigastric pain The diagnosis should be considered if serum amylase and lipase levels are markedly elevated

Hemorrhagic pancreatitis carries a mortality of approximately 50%, so treatment should be vigorous as soon as the diagnosis is made The essentials are continuous gastrointestinal suction replacement of fluid and electrolytes, and adequate analgesia Morphine should not be used, but meperidine (Demerol), 100 mg, with atropine, 0 4 mg subcutaneously every 4 hours as necessary, will usually relieve the pain Probantheline bromide (Pro-banthine), 30 mg every 6 hours intramuscularly, is also required When response to medical treatment is not prompt, surgical intervention may be life saving in the presence of pancreatic perforation, as in abscess formation or in bowel or gallbladder perforation

RED DEGENERATION IN A LEIOMYOMA

As the uterus enlarges in pregnancy, it is not uncommon for the blood supply to leiomyomas to diminish or to be interrupted The tumors undergo red degeneration, which may be associated with moderate pain The area of the

fibroids is tender. The treatment should be bed rest and analgesics Very rarely is myomectomy required during pregnancy, this is a very bloody operation and should be avoided

TRAUMATIC INJURIES IN PREGNANCY

Trauma to the gravid uterus can affect the mother and the fetus quite profoundly The same principles for diagnosis and management apply as in any trauma victim, but the potential complications and the margins of safety in the treatment of these patients are considerably altered For these reasons, the management of these patients must be undertaken by a team including the obstetrician, pediatrician, and appropriate medical surgical specialists However, one team member—preferably the obstetrician—should have primary responsibility for the care of the patient All patients with penetrating injuries should have toxin-antitoxin or toxoid, as well as adequate blood and fluid replacement, appropriate antibiotic coverage is essential if sepsis is present

ABDOMINAL TRAUMA

In blunt and penetrating injuries, the diagnosis is established by 1) history, 2) physical examination, 3) abdominal radiographs (flat film, upright, and left lateral decubitus), 4) examination of wounds, 5) paracentesis or culdocentesis and 6) laboratory studies Abdominal wounds should not be probed with instruments unnecessarily Such action may further contaminate the wound and cause further bleeding If examination fails to confirm that the peritoneal cavity has been entered, injection of Hypaque into the wound through a small rubber catheter may confirm the diagnosis on x-ray films Abdominal paracentesis or culdocentesis should be performed with a 10-cc syringe and an 18 gauge spinal needle In late pregnancy culdocentesis should not be done and paracentesis should be confined to the upper quadrants of the abdomen to avoid puncturing the uterus In early pregnancy, four quadrant taps may be helpful Laboratory studies should include CBC, typing and crossmatching, serum electrolytes, BUN, blood sugar, and serum amylase and lipase

Management

Laparotomy is mandatory if there is any evidence of intraabdominal hemorrhage, a ruptured viscus, or an enlarging retroperitoneal hematoma If none of these is suspected, the patient should be kept under careful observation, and the vital signs, fetal heart tones, and urinary output should be monitored every hour, more often if changing Serial pelvic and abdominal examinations should be conducted and serial laboratory studies carried out When operation is decided upon, the following points should be kept in mind 1) The incision should be in the midline to allow exploration of the entire abdomen 2) Uterine injury will require evacuation of the uterus and then repair or removal (see the section on the ruptured uterus in Ch 8, Hemorrhage in Late Pregnancy) Cesarean section should be carried out only for obstetric reasons If there is no evidence of uterine rupture present, the possibility that the abdominal pain

is associated with abruptio placentae should be considered If the diagnosis of uterine rupture is established at laparotomy, the uterus is best emptied by cesarean section even if the baby is premature Postmortem cesarean section is best reserved for cases of sudden maternal death in late pregnancy 3) Damage to other abdominal viscera should be managed in the usual fashion and will not usually require evacuation of the uterus If bowel contents have been spilled, operative procedures on the adnexa or uterus should be avoided if possible 4) Retroperitoneal hematoma is often associated with pelvic fractures The hematoma is best managed conservatively if it is limited Evacuation of the uterus may be required for exposure, to secure hemostasis by hypogastric artery ligation Pelvic packing may be necessary following such a ligation for complete control of bleeding If a hematoma has been present for over 6 hours and has been enlarging it may be extremely difficult to identify anatomic structures, and such normally simple procedures as hysterectomy may be difficult because of the abolition of normal landmarks

PELVIC TRAUMA

All patients with evidence of trauma should have careful evaluation of the lower urinary and gastrointestinal tracts for injury

Vulvovaginal Injuries

These are diagnosed by pelvic examination which should usually be performed under general anesthesia for accurate assessment

The treatment depends upon the findings Penetration of the cul-de-sac calls for immediate exploratory laparotomy to rule out injury to the uterus or other intraabdominal viscera Foreign bodies are removed if found Debridement, irrigation, and closure of wounds is important Incision and drainage of hematomas with ligation of bleeding points and appropriate placement of drains should be carried out Vaginal packing for tamponade is sometimes useful Tetanus prophylaxis or passive immunization with antitoxin is indicated, as for abdominal penetrating wounds Antibiotic therapy with ampicillin 1–2 g every 4 hours, clindamycin 600 mg every 8 hours and gentamycin, 80 mg every 8 hours, should be given intravenously With regard to delivery, in most instances, injuries will heal rapidly and vaginal delivery can be conducted *Cesarean section is indicated when lacerations are extensive and healing is* poor, or in any case in which there has been a recent repair of a lower urinary tract injury or gastrointestinal injury that communicated with the vagina.

PELVIC FRACTURES

The incidence of secondary injuries, the amount of blood loss, and the orthopedic and obstetric management will all be influenced by the forces causing the fracture the number of breaks in the pelvic girdle, and the degree of displacement of the fragments The diagnosis should be as precise as possible Orthopedic examination should be conducted to assess the fractures Diagnostic x-rays and additional studies should be carried out to determine the extent of injury to the urinary or gastrointestinal tracts

Management

Treatment depends upon the stage of pregnancy If the pregnancy has advanced 36 weeks or more, treat and plan for vaginal delivery except in the following circumstances 1) the orthopedic fixation and immobilization of the pelvic fracture would be disrupted by vaginal delivery in the near future, or 2) indications for exploratory laparotomy are present In these instances cesarean section combined with exploratory laparotomy is indicated

If the pregnancy is less than 36 weeks and there is no indication for exploratory laparotomy, 1) treat the fracture, 2) treat premature labor, if it develops, with an agent such as Ritodrine or orciprenaline, 3) deliver the patient vaginally at or near term unless there are obstetric, orthopedic, or urologic indications to perform cesarean section When cesarean section is indicated, orthopedic applicances may be applied at the same time This possibility should be kept in mind when the case is discussed with the consulting orthopedic surgeon

CARDIAC SURGERY

Any cardiac dysfunction great enough to cause cyanosis results in a high prematurity rate With the gradually increasing range of cardiac deformities, both congenital and acquired, that are coming under the corrective skill of the cardiac surgeon, it is advisable that elective surgery be performed prior to the onset of pregnancy In the event that cardiac surgery becomes indicated during pregnancy, however, it should be carried out The results are frequently surprisingly good

ADRENAL DISEASE

Hemiadrenalectomy for a hemorrhagic adrenal cyst in a pregnant woman was reported by Gillespie and McPhail (1962) with subsequent delivery of a term baby

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Preventable Maternal Deaths

Ralph E. Woods

Against stupidity the very gods
Themselves contend in vain
Johann Christoph Friedrich von Schiller (1759–1805)
The Maid of Orleans Act III Scene 6

In 1917, the Committee on Public Health Relations of the New York Academy of Medicine became interested in the problem of puerperal maternal mortality This interest derived from the fact that while death rates from other preventable causes had been steadily declining, those from puerperal causes had remained stationary

By 1920, the National Committee on Maternal Welfare had been established Its aims were to encourage the analysis of maternal deaths in every area of the United States and to try to prevent them by education of the laity, the hospitals, the community, and also the medical profession

Mechanisms for accomplishing this evolved over the next few years Williams of Philadelphia suggested that maternal mortality could be reduced only if each maternal death were thoroughly investigated, the responsibility of the parties involved ascertained, and the possibility of preventability carefully evaluated In 1930, he was appointed chairman of the Philadelphia Committee on Maternal Welfare, a function of the county medical society A significant stimulus toward the establishment of the committee was the evidence of discrepancies between the statistics for maternal deaths from the Philadelphia Bureau of Vital Statistics and those from the U S Bureau of the Census

In 1931 Williams began to hold open meetings at the Philadelphia General Hospital to review maternal deaths The physician in charge presented the case, facing the audience He had to reply to the questions and criticisms that were raised by its members All events that led to the maternal death were fully discussed, and no errors of commission or omission were overlooked Everyone was free to express his ideas The purpose was educational and not punitive, even though the atmosphere in which these meetings were conducted resembled that of a court trial

During the next 9 years, this analytic approach was pursued

As a result of case report analysis, a high percentage of preventable factors was identified On the basis of summary reports, corrective measures were undertaken These were essentially at the levels of physicians' care and community resources As a consequence, maternal mortality in Philadelphia dropped from 680/100,000 live births in 1931 to 230/100,000 live births in 1940 This improvement was obtained before sulfa drugs, antibiotics and blood bank services became generally available

This concept and activity has become widespread Study committees of some sort—local, regional, or state—now exist in every state Counterparts exist in other countries, in some cases at the national level Their continuing objective is the reduction of maternal mortality to an irreducible minimum by means of the principles and functions described above It is of interest that they have no actual legal power

The International Classification of Diseases (ICD)

At the present time the information on death certificates is codified according to the ICD, adapted in 1965 Maternal deaths are categorized under classifications 630–678 as shown below

Complications of pregnancy, childbirth and the puerperium
Ectopic pregnancy
Toxemias of pregnancy and the puerperium except abortion with toxemia
Hemorrhage of pregnancy and childbirth
Abortion
  Induced for legal reasons
  Induced for other reasons
  Spontaneous abortions
  Other and unspecified
Sepsis of childbirth and the puerperium
All other complications of pregnancy, childbirth, and the puerperium
Delivery without mention of complication

Maternal Deaths

Maternal death rates are computed per 100 000 live births There is some difference in the definitions of what constitutes a maternal death Maternal death is usually defined as the death of any woman while pregnant or within 42 days of termination of pregnancy, irrespective of the duration and site of the pregnancy Deaths within the 42-day time period are further subclassified

Period I, 1–7 days following termination
Period II, 8–42 days following termination

The World Health Organization defines a maternal death ' as a woman dying of any cause whatsoever while pregnant, or within 90 days of termination of pregnancy, irrespective of duration of the pregnancy, at the time of termination or the method by which it is terminated "

Efficient and accurate function of maternal mortality study committees depends heavily on the data collection system used for the compilation of vital

statistics Differences in terminology, deficiencies in completion, and inaccuracies in death certificates cause a number of difficulties Experience in the United States suggests that when reliance is placed on death certificates alone, a substantial number of maternal deaths are missed Presumably, the same is true in other countries Thus, it is difficult to develop accurate comparative statistics at regional, national, or international levels

In the United States when a maternal death is identified, the information is forwarded to a maternal mortality study committee Since the function of these committees is to identify problem areas in the causation of maternal deaths, the case is individually analyzed and categorized as to the cause of death, relationship to pregnancy, and the presence of avoidable factors

Case Study Analysis of Maternal Deaths

1. Nonobstetric maternal death (for example, an airplane accident)
2. Obstetric maternal death

   Direct Death resulting from obstetric complications of the pregnancy state, labor, or the puerperium, and from interventions, omissions, incorrect treatment or judgment of interrelated events resulting from any of the foregoing causes (for example, exsanguination due to a ruptured uterus following oxytocin stimulation)

   Indirect Death resulting from previously existing disease or a disease that developed during pregnancy, labor, or the puerperium but which was aggravated by pregnancy (for example, diabetes mellitus)
3. Preventability
   - Nonpreventable
   - Preventable
4. Responsibility
   - Physician
   - Patient and family
   - Community

This approach allows the identification of various weaknesses in the health care delivery system The tabulated information is intended primarily as an educational tool It is hoped that it stimulates corrective action if this is indicated

Table 16-1 shows the gross maternal mortality per 100,000 live births in the United States in 1975 The rate in the black population is three to four times as great as in the white population Cause specific rates are highest for the category "all other complications" (3 9 per 100,000 live births) Toxemia and sepsis each contributed 2 4 deaths per 100,000 live births Hemorrhage was the listed cause of death in 1 5 women per 100 000 live births Abortion and abortion related problems were responsible for 0 9 deaths per 100,000 live births

In the United States maternal mortality has declined steadily since 1930 The annual rate of decrease was relatively great until about 1950, when slowing occurred The downward trend has continued, however, so that in 1975, only 403 women died as a result of pregnancy or its complications

During this same period (1930–1975) numerous social changes have occurred in addition to major developments in medical practice Some of these have undoubtedly influenced this decline in the death rate Direct causal rela-

TABLE 16-1. Maternal Deaths per 100,000 Live Births and Maternal Mortality for Selected Causes, by Color, United States, 1975

Cause of death	ICD* classification	No. of deaths			Mortality/100,000		
		Total	White	All other	Total	White	All other
Ectopic pregnancy	631	50	19	31	1 6	0 7	3 2
Toxemia of pregnancy and the puerperium except abortion with toxemia	636-639	77	45	32	2 4	1 8	5 4
Hemorrhage of pregnancy and childbirth	632, 651-653	48	28	20	1 5	1 1	3 4
Abortion	640-645	27	11	16	0 9	0 4	2 7
Induced for legal indications	640 641	10	4	6	0 3	0 2	1 0
Induced for other reasons	642	4	1	3	0 1	0 0	0 5
Spontaneous	643	3	1	2	0 1	0 0	0 3
Other and unspecified	644 645	10	5	5	0 3	0 2	0 8
Sepsis of childbirth and the puerperium	670, 671, 673	76	51	25	2 4	2 0	4 2
All other complications of pregnancy childbirth and the puerperium	630, 633-635 654-662 672, 674-676	124	77	47	3 9	3 0	7 9
Delivery without mention of complications	650	1		1	0 0		0 2

*ICD, International Classification of Diseases Maternal deaths are those assigned to Complications of pregnancy, childbirth and the puerperium, category numbers 650-678, of the Eighth Revision, International Classification of Diseases, Adapted, 1965

(Based on Vital Statistics Report Vol 25, No 11, Supplement Feb 11, 1977)

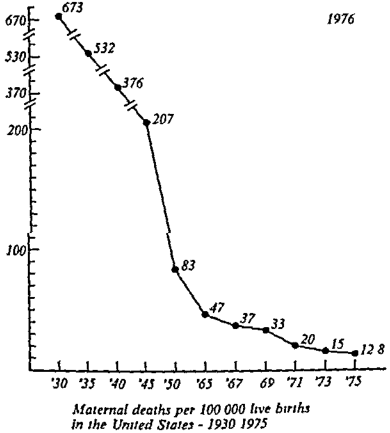
tionships are difficult to establish For example, in the 1930s and 1940s a substantial decline in maternal deaths occurred despite the general unavailability of antibiotics and blood transfusions

The following criteria should be used in judging whether observed relationships are causally related (Susser) The *time relationship* of the events must be correct, that is, one should follow the other closely, the association must be *consistent* on replication, the *specificity* and *strength* of the association should be consistent with a causal hypothesis, and a coherent explanation, in the light of current knowledge, should be possible

Judged by these criteria, the establishment of maternal mortality review committees, and the custom of delivering babies in well equipped and adequately-staffed hospitals appears to be causally related to the decline in maternal mortality The roles of changes in age and parity in reducing mortality have also been shown Other social changes and medical innovations have undoubtedly had similar beneficial effects

1. The causes of the decline in maternal mortality in the United States (Figure 16-1) must be carefully considered Caution must be used in extrapolating trends The persistence of deaths due to these causes may be due to "too little and too late" on the part of either health care provider or consumer

FIG 16-1 Maternal deaths per 100 000 live births in the United States from 1930 to 1975



They indicate the need for continued education for patients, doctors, and nurses

2. Preventable deaths have traditionally been charged to deficiencies in one of three areas physician care, patient actions, and community resources In a given case, two or more factors may be interrelated Individual case analysis has been shown to be an effective means of determining this It is also an effective way of ensuring quality medical care It must continue

   The decline in the total number of deaths regionally as well as nationally suggests that regional pooling of data may be needed to establish a data base adequate for determination of cause-specific mortality Alternatively, the issuing of biennial or triennial reports instead of annual reports might be considered Either approach might prove to be more educationally effective because the increased numbers would allow a more valid statistical analysis

3. Moreover, the maternal mortality review committees must begin to think in broader terms if they are to maintain educational effectiveness They must begin to function as perinatal study committees in an effort to improve pregnancy outcome for the mother and also for the infant Accumulation and analysis of pertinent data by such study committees might be very helpful in the redesign of health-care delivery services to overcome deficiencies in the areas of patient participation and use of community resources Such a program would provide a continued means of quality control of obstetric practices, both as to mortality and morbidity

4. In less well developed countries, and even in areas of the United States where services are unevenly distributed consideration should be given to restructuring the system of maternity care by the following steps

   A Establishment of peripheral maternal and child health centers closely linked with central hospital systems with rotating staff

   B. Screening for high risk pregnancies and appropriate referral

   C. Provision of efficient communication and transportation systems

   D. Use of mobile obstetric services

5. As social changes continue to evolve, it is essential that we find ways to alter staffing and practice patterns to meet the expectations of the consumers At one level, this may involve the integration of the nurse midwife and other health care providers into full membership on the obstetric team At another level, it may require accommodation within the limits of safety, to the desires of patients for a family centered obstetric experience

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Preventable Perinatal Mortality

Timothy C. F. O'Connor

There is no way in which humanity, patriotism or charity can be more laudably exerted or even a part of the public revenue more usefully employed than by enabling mothers to bring forth a healthy and hardy race of men

—W Buchan 1803

Perinatal mortality is best defined as any death occurring between 20 weeks' gestation and 28 days of age Using this definition the perinatal mortality rate for the United States in 1975 was approximately 22/1000 live births Before preventable perinatal mortality is discussed, it is necessary to evaluate the importance of various causes of death Autopsies are not always performed on stillbirths, so the precise cause of death is not always known Even if an autopsy is performed, the information that may be gathered from a macerated stillbirth is strictly limited Indeed, no cause is apparent for approximately 50% of stillbirths Because of the paucity of information available on fetal deaths, statisticians tend to concentrate on neonatal and infant death rates In this group, autopsies are more likely to be performed, but a glance at Tables 17-1 and 17-2 will demonstrate that two of the more common causes of death are described vaguely as "unspecified" or "unqualified" Although the relative importance of the common causes of death seen at necropsy (Table 17-3), may have changed since this study was made, it is likely that the five factors listed are still the most common causes of perinatal mortality These five factors are discussed here briefly, along with the possibility of prevention of each of them

PREMATURITY

The prevention of prematurity as a major cause of perinatal mortality would be greatly enhanced by the following

1. **The development of a consistently efficient method of inhibiting premature labor.** None of the substances available now can be truly said to do so
2. **The development of a method of accelerating fetal lung maturity.** Steroid treatment prior to delivery appears to

TABLE 17-1 Infant Mortality by Selected Causes United States, 1975

Cause of death	No./1000 live births
Congenital anomalies	2.8
Asphyxia of newborn unspecified	1 3
Immaturity unqualified	1 4
Influenza and pneumonia	0 7
Birth injuries	0 6
Certain gastrointestinal diseases	0 3
Other diseases of early infancy	5 4
All other causes	3 8
Total	16 1
Total deaths under 28 days	11 8

(Monthly Vital Statistics 24 (13) 9 1975)

TABLE 17 2. Neonatal Deaths (Under 28 days) by Selected Causes State of Missouri, 1975

Cause of death	No	% of total	Rate/1000 live births
Congenital anomalies	130	16 1	1 90
Asphyxia of newborn unspecified	110	13 6	1 6
Immaturity unqualified	66	8 2	0 96
Influenza and pneumonia	11	1 4	0 16
Birth injuries	42	5 2	0 6
Certain gastroentestial diseases	8	1 0	0 1
Other diseases of early infancy	371	45 9	5 4
All other causes	70	8 7	1 0
Total	808	100	11 8

(Missouri Vital Statistics Missouri Center for Health Statistics 1975 p 45)

TABLE 17 3 Main Findings at Necropsy

	Deaths	
Necropsy findings	No	%
Intrapartum hypoxia and/or birth trauma	586	29 8
Antepartum death with hypoxia or no lesion	419	20 95
First week deaths with respiratory distress	408	20 4
Congenital malformation	221	11 05
Infection pulmonary and extrapulmonary	126	6 3
Total perinatal deaths	2000	

(Attwood Stewart 1968)

have much to recommend it, but it must be given 24–48 hours prior to delivery, it is contraindicated in toxemia of pregnancy, and animal experiments suggest that it may have a deleterious effect on subsequent brain development (although the animal's brain development differs from that of humans, and the doses of steroid used were much larger than those used in humans)

3. **Intensive care of small babies during labor and after delivery.** Reports of very high mortality and a high incidence of mental handicap in surviving infants, together with gloomy prognostications that an increase in mental handicaps would result from efforts to increase survival rates, led to the development of an attitude of fatalism among obstetricians and pediatricians to the very small (<1500 g) infant (Lubchenco *et al*, 1963, Holt, 1970) However, recent reports indicate that when these infants are cared for in perinatal centers, with aggressive management of intrapartum complications as well as intensive neonatal care, not only is the survival rate increased, but the incidence of mental handicap is not nearly as high as was feared (Rawlings *et al*, 1971, Lubchenco *et al*, 1974) Thus, the time seems ripe for a review of this attitude of fatalism and its replacement with a resolve on the part of the obstetrician to ensure that these infants be delivered in optimum condition and in optimal surroundings This means delivery of these very small infants where neonatal intensive care is available, and the transfer of mother and fetus to a unit where such care is available, if necessary and possible It also entails monitoring these fetuses during labor with the more liberal use of cesarean section for fetal distress and malpresentations

INTRAPARTUM HYPOXIA AND/OR BIRTH TRAUMA

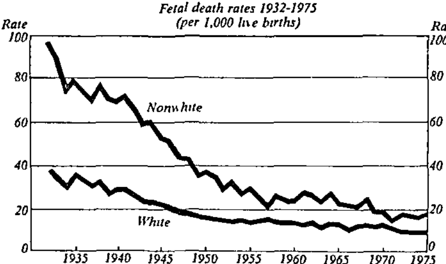
The widely used technique of fetal heart rate monitoring is designed to detect early evidence of intrapartum hypoxia, but monitoring cannot, alas, be said to have any significant impact on perinatal mortality (Renov, 1975, Goodlin, 1977) The importance of using some other monitoring technique (fetal scalp sampling) as well as fetal heart rate monitoring has already been stressed (see Ch 9, Intrapartum Emergencies), but it is conceivable that some quite different method of monitoring may replace either or both of these

Birth trauma usually follows precipitate or prolonged labor, breech delivery, or instrumental delivery Heroic efforts to achieve vaginal delivery at all costs have become relics of the past, and judicious acceleration of labor has virtually eliminated prolonged labor One of the common indications for midforceps delivery is the passage of an arbitrary period of time in the second stage of labor. Recent evidence (Cohen, 1977) suggests that such intervention may not be necessary, provided there is no evidence of fetal distress This study should make the obstetrician review the indications for midforceps delivery before performing a potentially dangerous operation

LATE FETAL DEATHS (STILLBIRTHS)

The fetal death rate has not continued to decrease at the same rate as the infant mortality rate in recent years (Fig 17-1; Table 17-4) The concept of selection of patients at high risk for fetal loss by virtue of factors

FIG 17-1 A. The percentage of decrease in fetal deaths has been maintained in recent years (Missouri State Board of Health, Vital Statistics Reports, 1932–34 and 1937–48, U S Bureau of the Census, Mortality Statistics, 1935–36, Missouri Division of Health, Vital Statistics Reports, 1949–date, 1932–44 are recorded data, and 1945 to date resident.) B. The decline in infant deaths continued up to 1975 (Missouri State Board of Health, Vital Statistics Reports, 1932–34 and 1937–48, U S Bureau of the Census, Mortality Statistics, 1935–36, Missouri Division of Health, Vital Statistics Reports, 1949 to date 1932–44 are recorded data, and 1945 to date resident)



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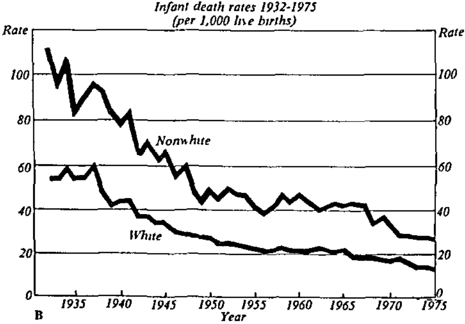

B

TABLE 17.4 Fetal and Infant Death Trends, 1969–1975

Year	Fetal deaths/1000 Total	Fetal deaths/1000 White	Fetal deaths/1000 Nonwhite	Infant deaths/1000 Total	Infant deaths/1000 White	Infant deaths/1000 Nonwhite
1969	13.2	12.1	19.6	21.3	18.6	36.4
1970	13.8	12.7	19.8	19.5	17.0	33.6
1971	12.5	11.8	16.2	20.2	18.6	28.0
1972	12.1	10.9	18.2	18.3	16.2	28.5
1973	11.8	10.6	17.8	17.7	15.9	27.1
1974	12.2	11.2	17.3	16.9	15.0	17.0
1975	11.5	10.0	19.3	16.4	14.5	25.9

(Missouri Division of Health Vital Statistics Reports resident data)

such as maternal age, parity, medical disease, socioeconomic status, and past obstetric performance, together with referral of such patients to perinatal centers may be expected to prove useful in preventing this group of perinatal deaths. Unfortunately, most of the complicated schemes for selecting these high-risk pregnancies identify approximately 30% of the population as "high risk," while only 6–10% of infants actually need intensive care (Ryan and Butterfield, 1975). This imperfection in selection has prevented many obstetricians from referring potential problem pregnancies.

CONGENITAL MALFORMATIONS

Until the cause of a congenital malformation is known, it cannot be prevented. Amniocentesis and genetic counseling will be very helpful in many cases. Many congenital malformations can be diagnosed *in utero*, and early diagnosis of some of these may suggest arrangements for delivery in a unit where pediatric surgery is immediately available.

INFECTION

An awareness on the part of the obstetrician of the significance of prolonged rupture of the membranes and prolonged labor is essential to the prevention of this group of deaths (see Ch. 4, Life-Threatening Infections, and Ch. 12, Emergencies in the Newborn).

There is accumulating evidence that neonatal intensive care units improve the outcome for sick infants (Carrier *et al.*, 1972). The nearer to such a unit the infant can be delivered, the fewer problems with transfer that will arise. However, the obstetrician is naturally reluctant to refer all his "high-risk" patients to an anonymous unit, but would be much happier if he could attend his patient in such a unit himself. The solution appears to be open staffing for all such units.

The information that may be obtained from an autopsy performed by a skilled neonatal pathologist in the case of a stillbirth or neonatal death is often

very valuable, and such examinations should be performed, where possible, in an effort to highlight preventable causes of fetal and neonatal mortality

As perinatal mortality continues to fall, obstetricians and pediatricians are concentrating more on neonatal morbidity—particularly long term morbidity—and its relation to obstetric and pediatric practice Such statistics take many years to collect and continuous follow up of any infant delivered after a complicated pregnancy, labor, or delivery is essential for collection of these very important data

As pointed out by Correy (1977), with changing trends in the practice of obstetrics prospective studies are necessary to evaluate the effects of these trends on perinatal mortality and morbidity For this reason the use of a simple, universally applicable Obstetric Record should be encouraged internationally by the World Health Organization (W H O) An even more important initial step would be the standardization of definitions by W H O to ensure that accurate comparisons can be made from one country to another Until this is done the use of perinatal mortality as an index of maternal and infant care is grossly misleading

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Appendix

CESAREAN SECTION INSTRUMENTS

9 sponge-holding (ring) forceps
3 needle holders
2 scalpel handles (Nos 4, 7, and 3) with fresh blades available
2 Parker retractors
2 small tissue forceps with teeth
2 large tissue forceps with teeth
2 small tissue forceps without teeth
2 large tissue forceps without teeth
2 small Deaver retractors
2 large Deaver retractors
2 small Richardson retractors
2 large Richardson retractors
1 large uterine elevator
1 straight suction tip
1 tonsil suction tip
1 obstetrical forceps (Elliot type)
1 narrow malleable ribbon retractor
1 wide malleable ribbon retractor
2 single tooth tenaculum forceps
2 double tooth tenaculum forceps
2 small Babcock clamps
2 large Babcock clamps
4 medium sized straight Kocher clamps
6 medium sized curved Kocher clamps
4 heavy clamps
12 towel clips
6 small Allis tissue forceps
4 large Allis tissue forceps
6 Pean clamps
6 medium curved Kelly clamps
4 Moynihan clamps
12 small curved hemostats
12 small straight hemostats
6 Thompkinson clamps
1 De Lee retractor (left, right, and middle blades)
2 suture scissors
2 curved Mayo scissors
2 long pelvic scissors
6 laparotomy packs with metal rings attached
2 basins with saline for moistening packs & washing off gloves

SUTURE MATERIALS

3-0 cotton on a reel for tying small blood vessels
2-0 cotton for fascial closure
2 0 chromic catgut on an atraumatic needle for the peritoneum
1-0 chromic catgut on an Atroloc needle for two layer closure of the uterus
Metal skin clips

INSTRUMENTS FOR VAGINAL DELIVERY

1 pair of obstetrical forceps
4 towel clips
2 tissue forceps with teeth
4 Allis tissue forceps
2 pairs of straight scissors
6 small curved hemostats
1 needle holder
2 tubes of 1 0 chromic catgut with Atroloc needles
2 tubes 2-0 chromic catgut with atraumatic needles

1 straight needle
2 right angled vaginal retractors
2 sponge holding (ring) forceps for inspection of the cervix
2 uterine dressing forceps
1 No 16 French rubber catheter
1 urine collection bottle
1 soft rubber bulb syringe for aspiration of the baby's oropharynx
Version gloves need not be on the set but should be immediately available for manual removal of the placenta or internal version

LINEN FOR VAGINAL DELIVERY

1 pair of leggings with retaining rings
8 towels
1 rubber sheet for placement under the patient
2 abdominal drapes
3 gowns
3 caps
3 masks
2 umbilical dressings with tapes or clips
20 gauze squares
2 tagged vaginal packs for supporting the uterus during perineal repair
2 five yard gauze rolls
6 pairs of rubber gloves
2 perineal pads
1 sterile sheet for the baby's table
2 sterile blankets for the baby

DRUGS AVAILABLE IN DELIVERY ROOM

A For preparation of the perineum and catheterization, aqueous pHisohex or other suitable antiseptic solution should be available
B *Drugs*
Oxytocin (Pitocin) ampules
Methylergonovine maleate (Methergine) ampules
Ergonovine maleate (Ergotrate) ampules
Epinephrine (Adrenalin) 1 1 000 sol in ampules
Atropine sulfate
Morphine sulfate, 2 intravenous infusion sets with No 18 gauge needles
Dextrose 5% in water 1,000 ml
Isotonic saline 1,000 ml
Dextran 500 ml
Hypodermic syringe with needles
Procaine 1% solution
Ether and a mask for administration by the open method
Trichlorethylene with a hand inhaler for self administration by the patient
Fresh silver nitrate 1% for baby's eyes
Naloxone (Narcan) adult and infant

Index